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## THE TREATMENT OF BURNS AND SCALDS

### WITH ESPECIAL REFERENCE TO THE USE OF TANNIC ACID *

BY PHILIP H. MITCHINER, M.D., M.S. LOND., F.R.C.S. ENG.,

HON. SURGEON TO THE KING; SURGEON WITH CHARGE OF THE SEPTIC WARDS AND SENIOR SURGEON TO OUT-PATIENTS, ST. THOMAS'S HOSPITAL, LONDON

IN dealing with any subject in surgery it is safe to say that nothing new is likely to be revealed, and research into the subject of the treatment of burns has merely proved this well-worn aphorism; for we find that while in Europe Hippocrates, in 435 B.C., was advocating the treatment of burns by a mixture of bees-wax with myrrh and certain balsams of an antiseptic nature—a treatment which has persisted in modified forms in Europe throughout the centuries—yet a far older treatment advocated some 5000 years ago by the physicians in China and carried out pretty universally in that country to this day—the treatment of burns by tannic acid in the form of tea—has been revivified by the Americans and reached us from the West instead of from the East.

In this paper an effort has been made to correlate the results of the treatment of burns and scalds in St. Thomas's Hospital, over equal periods, under various treatments, and to endeavour to compare their success as judged by the mortality in those more severe cases which have been admitted to the hospital wards for treatment.

### Factors Affecting the Mortality of Burns

Before dealing with the mortality of burns and scalds, there are two important factors to be considered irrespective of the method of treatment used.

The first of these, as pointed out by Donald,[1] is that the improved social conditions in the homes of the poor, and the disappearance of the open light and open fire have done much to diminish the number and severity of burns coming for treatment to the great hospitals of the metropolis. Taking the figures from 1899–1903 at the London Hospital, he has shown that the mortality for the 999 burns and scalds admitted during those years was 25·3 per cent., whereas in 1924–28 the number of admissions had diminished to 286, or rather less than one-third, and the mortality had dropped to 6·6 per cent., although the treatment used was practically the same during both these periods. This he attributes largely to the passing of the Children's Act in 1908, which made it a punishable offence to leave a child alone in a room containing an open fire grate insufficiently protected, and he pointed out that in the years 1909–13, immediately following the passing of this Act into law, the number of burns admitted to hospital diminished by 50 per cent.

At St. Thomas's Hospital the figures are by no means so conclusive, for though the number of burns and scalds treated as out-patients is slightly smaller, the number of burns and scalds admitted to the hospital has remained very steady throughout a number of years, both before and after the passing of the Children's Act in 1908, and has been approximately 60 per annum from the year 1894 onwards until 1930, since when the numbers have risen to about 70 per annum. It is, however, of interest to find that whereas in the years before the passing of the Children's Act the number of burns to scalds averaged 3 to 2, now the proportion of burns to scalds has reversed to 1 to 2·5, while in children under 10 years of age it is approximately 1 burn to 7 scalds. Donald's figures give the ratio as having changed from 1 burn to 1·5 scalds to 1 burn to 2·3 scalds in children under 6 years in the same period. This is a complete reversal of the type of injury and, as we shall see in dealing with the next point, has a great and beneficial influence on the mortality irrespective of the treatment used.

The second point which must be considered is one which has not, I think, been sufficiently realised previously—viz., the very much smaller mortality from scalds than from burns. While we must admit that the introduction of the covered light and the covered fire and the passing of the Children's Act have diminished the number of burns considerably, we find that this has not been so with the number of scalds which has risen considerably in proportion—i.e., from 2 scalds to 3 burns in 1899, to 2·5 scalds to 1 burn in 1929 (in children under 10 years 7 scalds to 1 burn 1929). From Table I. it will be seen

TABLE I.—*Mortality of Burns and Scalds in St. Thomas's Hospital* (1894–1932).

| Year group. | Treatment. | Mortality (per cent.). | | Burns and scalds. |
|---|---|---|---|---|
| | | Burns. | Scalds. | Combined figure (per cent.). |
| 1894–1898.. | Baths and ointment. | 39·6 | 18·8 | 30·8 |
| 1900–1903.. | 2 per cent. picric. | 39·7 | 7·0 | 25·8 |
| 1924–1928.. | 2 ,, ,, | 15·5 | 7·5 | 9·4 |
| 1929–1932.. | 2 ,, tannic. | 4·0 | 1·7 | 2·4 |
| Do. under 10 years | do. .. | 11·1 | 2·2 | 3·7 |

that whereas the average mortality for burns over the period of years from 1894–98 was 40 per cent., that for scalds for the same period was only 19 per cent. Again, during the period from 1900–1903 the average mortality from burns was 39·7 per cent., whereas from scalds it was only 7 per cent. Here it is of interest to note that in 1899 (Table II.) picric acid was used for burns and the mortality fell to 33 per cent., but the mortality of scalds, still for some reason treated by old methods, was 40 per cent. Again, from 1924–28 (Table I.) the mortality for burns was 15·5 per cent., but for scalds only 7·5 per cent., and from 1929–32, inclusive, the mortality was, burns 4 per cent., scalds 1·7 per cent.

The Registrar-General's mortality figures are unfortunately not helpful, for they give the number of deaths per million of population, and offer no indication of the relative number of non-fatal burns and scalds. They show a greater number dying from burns than scalds—20·8 burns and 18 scalds per million, all ages. It is startling and unexpected to find that the female deaths from burns are just over twice as great as the males throughout the period (1924–31), while in the case of scalds the proportion is 2 females to 3 males. Further, whereas the number of burns to scalds in the first period (1894–98) was 275 burns to 202 scalds, in the second period (1900–1903) it was 239 burns to 172 scalds; and in the third

* Hunterian lecture delivered before the Royal College of Surgeons of England on Feb. 1st.

period (1924–31) it was 132 burns to 360 scalds; showing that the total number of burns and scalds admitted to St. Thomas's has remained very constant, yet there is a considerable increase in the number of scalds to burns—a factor the importance of which must be realised when considering the mortality of these cases apart from the method of treatment adopted, and one which I venture to point out has so far apparently escaped notice. It is, however, of some importance, for I find that whereas some writers by "burns" mean burns only, others include scalds under this heading, a proceeding which vitiates the value of comparative tables.

### Causes of Death from Burns

In considering the causes of death from burns, as they have been admirably summarised by Wilson,[2] we have the stage of initial shock (or, as I prefer to call it, "shock") which immediately follows the upset to the nervous system resulting from the exposure to flames or scalding fluid. He estimates the mortality in this period as only 2½ per cent. The experience at St. Thomas's Hospital places it even slightly lower in those cases which survive to be admitted to hospital, though Donald gives this figure as far higher and increasing from 30 per cent. (1899) to 58 per cent. (1928).

Secondly, the stage of acute toxæmia due to the absorption of toxic substances from the damaged tissue in the burnt or scalded area. Here the experience at St. Thomas's Hospital leads one to agree emphatically with Wilson's opinion that this occurs 6 to 24 hours subsequent to the injury, and not as stated by Fraser [3] on the fourth day or later. As has been shown by Davidson [4] and Fraser,[5] this toxæmia is the result of histamine freed into the blood from damaged tissues which possibly causes diminution of blood chloride, and is best combated, if and when it has occurred, by exsanguination transfusion as suggested by Ajello and Parascando,[6] and advocated and practised with such success by Fraser,[5] Harris,[7] and others. This method, which consists in removing a measured quantity of the toxic blood from the patient and replacing it by a somewhat larger volume of whole blood from a donor, is not necessary if by coagulation of the damaged tissues we can prevent absorption toxæmia. It is therefore unnecessary, as will be shown, in the treatment of burns the surface of which has been sufficiently coagulated with tannic acid.

There is, however, at an earlier period (6 to 12 hours) a very important factor which seems to have escaped the attention of Wilson, for he does not mention it. Attention is however devoted to this subject by Underhill,[8] who discovered that the loss of blood-serum from the burnt surface in the first hours after a burn is very excessive—a fact well known clinically and demonstrated both by the condition of the dressings on the burns and the extreme thirst and restlessness of the patient. Underhill showed that with rabbits this loss may reach 70 per cent. of the blood volume, that the maximum loss occurred with third degree burns of large extent, moreover that where one-sixth of the total surface is burnt death almost inevitably follows. In analogy, a man of 65 kg. body-weight who might be expected to have 5000 c.cm. of blood in his body would lose 3500 c.cm. of serum in the 24 hours following a third-degree burn, involving one-sixth of the total body surface, and the majority of this loss occurs in the first 12 hours. It will be seen therefore that this factor constitutes a very potent cause of that collapse which subsequently ensues and which is responsible for something like 80 per cent. of the deaths after burns. This collapse is due both to the toxic absorption and this extensive loss of fluid from the burnt area.

The excessive loss of serum may be combated by the administration of fluid intravenously, subcutaneously, and orally, but with an extensive burn it is next to impossible to get sufficient into the tissues to replace the loss, and it is essential therefore that some method of combating this loss, such as coagulation of the surface of the burnt area, shall be devised which will be sufficient, both mechanically and physically, to prevent the escape of fluid from the capillaries on the surface of the damaged tissues. It was, I think, here that the picric acid treatment constituted such an advance on the older bath and grease method, though it failed inasmuch as the coagulum did not penetrate the tissues to prevent the absorption of toxic substances from the deeper tissue damaged in the accident. Again, owing to the toxicity of the picric acid, manifested by skin rashes, irritation, and not infrequently by albuminuria, it was seldom possible to retain this dressing more than two or three days, after which paraffin had to be substituted and sepsis not infrequently supervened.

Thirdly, there is the stage of septic toxæmia which constituted so serious a problem in the few cases which in the old days survived the previous stage. This is not a serious problem if the injury is treated by picric acid, and is practically non-existent in the tannic acid treatment, where with proper and adequate preliminary cleansing of the damaged area sepsis is practically never seen.

### Reasons of Failure of Older Methods of Treatment

The practice at St. Thomas's Hospital before 1899 was to treat all burns by daily baths, with the object of washing away septic material, and applying an antiseptic grease dressing between these periods in an endeavour to obtain a painless dressing and a vain hope of preventing the suppuration which invariably occurred. But no local treatment was present either to prevent the absorption of collapse producing histamine bodies, or to stop the escape of serum from the damaged tissues. As will be seen from Table II., the average mortality during these years for burns was 39·6 per cent., and for scalds was 18·8 per cent. (compared with the Sick Children's Hospital, Toronto,

TABLE II.—*Mortality of Burns and Scalds in St. Thomas's Hospital* (1894–98).

*Treatment.*—Baths and boric ointment.

| Year. | Burns. | | | Scalds. | | |
|---|---|---|---|---|---|---|
| | No. | Deaths. | Per cent. | No. | Deaths. | Per cent. |
| 1894 .. .. | 67 | 19 | 28·0 | 46 | 10 | 21·0 |
| 1895 .. .. | 63 | 25 | 39·0 | 44 | 2 | 4·5 |
| 1896 .. .. | 48 | 26 | 54·0 | 34 | 11 | 32·0 |
| 1897 .. .. | 49 | 18 | 36·0 | 33 | 7 | 21·0 |
| 1898 .. .. | 48 | 21 | 43·75 | 45 | 8 | 18·0 |
| Total 5 years.. | 275 | 109 | 39·6 | 202 | 38 | 18·8 |
| 1899 .. .. | 45 | 15 | 33·0 | 30 | 12 | 40·0 |
| Treatment: | 2 per cent. aq. picric. | | | Baths and ointment. | | |

37·2 per cent., Harris,[7] and 35·4 per cent. at Edinburgh, Wilson [9]).

Following the introduction of the picric acid treatment for burns by, I believe, Sir Cuthbert Wallace in 1899, the mortality at St. Thomas's Hospital during the succeeding four years, as seen from Table IIIA., diminished considerably for scalds, showing an average for the period of 7 per cent., that of burns remaining at 39·7 per cent.; while in a corresponding five-year period immediately preceding the introduction of tannic acid the mortality for burns had fallen, I think partly owing to the introduction of adequate preliminary cleansing of the damaged area, partly to improved social conditions and the change in the proportion of burns to scalds (Table III.B) to 15·5 per cent., while that for scalds remained approximately the same at 7·5 per cent. No exsanguination transfusion was carried out on these cases.

TABLE III.—*Mortality of Burns and Scalds in St. Thomas's Hospital* (A, 1900–03; B, 1924–28).

*Treatment.*—2 per cent. aqueous picric acid for two or three days, then paraffin dressings in most cases.

A

| Year. | Burns. | | | Scalds. | | |
|---|---|---|---|---|---|---|
| | No. | Deaths. | Per cent. | No. | Deaths. | Per cent. |
| 1900 .. .. | 49 | 16 | 32·6 | 32 | 3 | 9·4 |
| 1901 .. .. | 55 | 29 | 52·7 | 42 | 1 | 2·3 |
| 1902 .. .. | 51 | 18 | 35·2 | 35 | 3 | 8·5 |
| 1903 .. .. | 39 | 14 | 35·8 | 33 | 3 | 9·0 |
| Total 4 years.. | 194 | 77 | 39·7 | 142 | 10 | 7·0 |

B

| | | | | | | |
|---|---|---|---|---|---|---|
| 1924 .. .. | 9 | 1 | 11·0 | 29 | 0 | 0 |
| 1925 .. .. | 13 | 4 | 30·8 | 39 | 3 | 7·7 |
| 1926 .. .. | 11 | 1 | 9·1 | 40 | 3 | 7·5 |
| 1927 .. .. | 14 | 2 | 14·3 | 52 | 5 | 9·6 |
| 1928*.. .. | 11 | 1 | 9·1 | 25 | 3 | 12·0 |
| Total 5 years.. | 58 | 9 | 15·5 | 185 | 14 | 7·5 |

* In addition, treated with 2 per cent. tannic spray, 7 burns and scalds with 1 death—14·2 per cent.

These figures compare very favourably both with the figure quoted by Wilson [9] for the Edinburgh Royal Infirmary (22·4 per cent.) during the same period, and the Sick Children's Hospital, Toronto (29·6 per cent. in 213 cases), where exsanguination transfusion was used combined with local treatment.

What then was the reason of this improvement in mortality where picric acid 2 per cent. aqueous solution was used unless that the coagulation of the surface protein both lessened collapse and stemmed the escape of serum?

## Considerations in Treatment

The treatment of burns and scalds therefore, following adequate cleansing of the damaged area, aims, in the main, at lessening the collapse by stopping the absorption of those bodies which cause this collapse and death in from 12 to 48 hours after the burn, and also in diminishing the loss of fluid from the burnt area which, as has been shown, is so serious and so often fatal in the first 6 to 12 hours.

A further aim is prevention of the toxæmia from absorption of septic material from the infected burnt area which may be manifested elsewhere, especially as septic broncho-pneumonia, and cause death in from 5 to 12 days.

The initial shock and suffocation cannot be affected by local treatment of the burnt area, and death from these causes can be ameliorated only by efficient first-aid treatment from those on the spot at the time of injury.

## Tannic Acid Treatment

When I was in Detroit in 1926 I was privileged to see Dr. Davidson's treatment of burns by compresses of tannic acid solution, and I was very much struck with the fit condition of the patients and the absence of sepsis resulting from this treatment. I was, however, convinced that a treatment which necessitated the removal of the protecting scab with the dressings at frequent intervals, with considerable pain to the patient, was not ideal.

On taking over the charge in November, 1928, of the septic wards at St. Thomas's Hospital, to which all the burns and scalds are admitted, I adopted the spray method advocated by Wilson,[2] saving that I found a 2 per cent. solution of tannic acid was adequate to secure good results. I was immediately struck by the enormous improvement in the general condition of the patients and the entire absence of pain and sepsis in the areas so treated. It was a noteworthy fact that patients with very extensive burns and children with scalds were comfortable, bright, and alert the day following the injury, read, talked cheerfully, and ate well, while the pulse was not rapid, nor the temperature raised.

TABLE IV.—*Mortality of Burns and Scalds in St. Thomas's Hospital* (1929–32).

*Treatment.*—2 per cent. tannic acid, 1/2000 mercuric chloride; spray *or* compress.

| Year. | Burns. | | | Scalds. | | |
|---|---|---|---|---|---|---|
| | No. | Deaths. | Per cent. | No. | Deaths. | Per cent. |
| 1929 .. .. | 12 | 0 | 0 | 27 | 0 | 0 |
| 1930 .. .. | 15 | 1 | 6·6 | 37 | 2 | 5·4 |
| 1931 .. .. | 21 | 1 | 4·7 | 53 | 1 | 1·8 |
| 1932 .. .. | 26 | 1 | 3·8 | 58 | 0 | 0 |
| Total 4 years.. | 74 | 3 | 4·0 | 175 | 3 | 1·7 |

It was obvious, however, that there were many disadvantages to this method of treatment, to wit, (*a*) the necessity for leaving the area exposed to the air while at the same time keeping it and the patient warmed; (*b*) the disadvantage of the mechanical restraint necessary to keep the damaged area at rest and free from interference and irritation: (*c*) the need for constant attention and repeated hourly sprayings for from 10 to 24 hours (and usually, in my experience, nearer the latter period of time); (*d*) the obvious impossibility of being able to spray and expose the entire burnt or scalded surface in those cases where the body had been burnt both in front and at the back; (*e*) and—a minor detail—the necessity for hourly making of fresh solutions. This last was necessary, as I discovered that with solutions kept after this time the crust produced was soft in consistency, light in colour, and though it gave an equally satisfactory result as when constant fresh solutions were applied, yet the patient suffered some pain and inconvenience which could be avoided under the rigid

black coagulum obtained with repeated applications of freshly made 2 per cent. tannic acid.

These facts made this method of treatment applicable only in the case of in-patients in large hospitals, and possibly the offspring of that diminishing class, the millionaire, and even in these cases only to those patients who were sufficiently thoughtful to be burnt on one side alone. I therefore set about to try to discover some other method which should be applicable universally to the treatment of burns and scalds in the out-patient departments, by general practitioners, and both at home and in works as a first-aid application. And here I was helped by the suggestion of a compress for first-aid in the Medical Research Council's report.[10] It struck me that if an antiseptic were added it would enable the solution to be made with clean tap water, and prevent possible infection from outside sources. I was further guided in the choice of an antiseptic by the knowledge that perchloride of mercury was a precipitant of proteins, and indeed, on inquiry, I found that Petit, and I believe also Lord Lister, had in the early part of the nineteenth century employed perchloride of mercury as a treatment for burns with the object of precipitating surface protein by an antiseptic and so preventing toxic absorption and local sepsis. Owing, however, to the toxic nature of the mercury solution all those cases had either died or suffered severely from mercurial poisoning, and this method of treatment had been abandoned. I found, however, that when added in a strength of more than 1/2000, the perchloride of mercury interfered with the coagulating

FIG. 1

Severe third and fourth degree burn. Ten hours after injury. Face sprayed; arms "compressed" (a clean bandage is applied over the stained compress and bandage).

action of the tannic acid and produced only a soft soapy crust, which was unsatisfactory and did not relieve the patient's pain or completely prevent collapse.

I therefore commenced in the autumn of 1929 to use in the out-patient department compresses soaked in 2 per cent. tannic acid and 1/2000 perchloride of mercury for the treatment of all minor burns and scalds. These were made of lint or gauze but I have found the latter more satisfactory, and

FIG. 2

The same patient 14 days later. Coagulum and compress removed.

at first this was re-wetted on the outside hourly for six or eight times with freshly made solution. This frequent damping was inconvenient with out-patients, even though I had tablets made for the patients to take home with them. Further investigation has shown this re-damping to be unnecessary to the success of this method, which has been for over three years in almost universal use in both the in- and out-patient departments of St. Thomas's Hospital, and latterly it has been in use by several general practitioners and as a first-aid dressing in various works, and in my own home, where it has proved satisfactory.

### CHANGES OF TANNIC ACID AND ITS SOLUTIONS ON KEEPING

The next thing that occurred to me was why a fresh solution was necessary hourly? On analysis of the tannic acid used at St. Thomas's Hospital, it proved to be a natural gallo-tannin prepared from Aleppo galls, and to contain when fresh 88 per cent. gallo-tannin and 6 per cent. gallic acid, and some moisture. Thanks to the aid of Prof. R. H. A. Plimmer and Mr. J. Lowndes, of our chemistry department, I soon knew that even at the end of two years no change had taken place in the colloidal state of the tannic acid solution, and it was necessary therefore to seek some other explanation as to why a stale solution gave an unsatisfactory crust. Frankly I have not been able to find it. In this investigation I have had the help of Mr. C. W. Peck, the pharmacist at St. Thomas's Hospital, and the great assistance of Mr. W. A. Woodward, one of the dispensers. Mr. Woodward, working by the colorimetric method advocated by Ainsworth Mitchell,[11] has shown that even after five years a 2 per cent. solution of tannic acid contains no more than 8·45 per cent. of gallic acid as a result of hydro-

lysis and 73 per cent. unaltered gallo-tannin, and that the percentage of gallic acid in solutions of earlier date is considerably less, I have been convinced that change to gallic acid does not occur to any appreciable extent. Moreover, this hydrolysis to gallic acid takes place extremely slowly in solutions free from moulds, which do not develop in those containing 1/2000 perchloride of mercury, and it has been shown that in a three-months-old 2 per cent. solution with 1/2000 perchloride of mercury no practical increase occurs in the percentage of gallic acid. Incidentally, on sterilising and using the five-year-old solution above referred to on a burn, as a compress, a good scab was obtained with a perfect result.

A further possible cause for failure of the stale solution was that owing to an increased density of colloid a solution of diminished strength only could get through the nozzle of the atomiser, but this was easily disproved. Then was it possible that oxidation was causing some change in the solution though none was demonstrable? A solution was charged with carbon dioxide to prevent this possible oxidation, and though this remained quite free from those traces of pyrogallol which cause the darkening of any tannic acid solution exposed to the air, the

FIG. 3

Slight scald of hand. Showing complete epithelialisation on removal of compress at end of 10 days.

crust produced was not in any way different to that obtained from using the ordinary stale solution of tannic acid, it was softer and lighter in colour.

It was obvious, therefore, that no apparent change occurred in the solution, and I considered that probably longer contact of the solution with the area should result in a satisfactory coagulum, and, as already stated, observations proved this surmise to be correct. It struck me when I found that the burnt surface, covered by a sterile gauze dressing, of a patient lying on a sheet soaked in the overflow from the tannic acid sprayed on the damaged and exposed area, formed a perfectly good crust, that herein lay a possible easy method of treatment. By the sacrifice of a soiled sheet I evolved the method of treating these burns and scalds with a compress left undisturbed on the previously cleaned area until the scab separated, usually in from 14 to 21 days (Figs. 1 and 2).

In order to prevent the growth of the moulds in the tannic acid solution, and so diminish the hydrolysis to gallic acid and at the same time to keep an antiseptic in the area and possibly so minimise the chance of sepsis from outside sources, I looked round for some suitable antiseptic to add to the 2 per cent. tannic acid solution. Here I was helped by previous experience and decided to use the 1/2000 perchloride of mercury, which again proved quite efficient in inhibiting growth of moulds and so reducing hydrolysis to a minimum, while it also acted as an efficient antiseptic in the coagulum and so prevented outside infection. I again found that when added in a strength of more than 1/2000 the perchloride interfered with the coagulating action of the tannic acid and produced a soapy crust which was unsatisfactory and did not relieve the patient's pain.

### TECHNIQUE OF THE COMPRESS METHOD

A compress of 2 per cent. tannic acid with 1/2000 perchloride of mercury makes a most efficient first-aid dressing for all burns and scalds, as I have proved in my own home. But in the home and other places where burns occur but rarely, tablets or powder, to be dissolved in warm water and applied by soaking clean linen in the resulting solution, will be found more advantageous than keeping a large quantity of stock 2 per cent. solution.

The formulæ for the tablets and powder are as follows:—

*Powder:* tannic acid, grs. 17½; perchloride of mercury, gr. ½; to be dissolved in 2 oz. of warm water, when a 2 per cent. solution will result.

*Tablets:* tannic acid, grs. 17½; perchloride of mercury, gr. ½; boric acid, gr. 1; to be dissolved in 2 oz. of warm water, when a 2 per cent. solution will result.

Whatever method of first-aid treatment may have been employed—and I hope as time goes on the method I am about to describe may become universally adopted as a first-aid dressing—it is essential that this first-aid dressing, even if of tannic acid, should be removed, which it can be most easily, and the area carefully cleaned before the final tannic acid dressing is applied. By this careful cleansing alone can subsequent sepsis be avoided; and here let me say that should sepsis supervene during a course of treatment, it is only necessary to remove the tannic acid dressing, re-clean the area thoroughly, and

TABLE V.—*Opium Dosage for Cleansing Burns and Scalds*

I.—*In Children*

| Age. | Preparation. | Dose. |
|---|---|---|
| 1/12 .. .. | Tinct. camph. co. | ♏ ii.–iii. |
| 2/12 .. .. | „ „ „ | ♏ iv.–vi. |
| 3/12 .. .. | Tinct. opii. | ♏ ¼–½. |
| 6/12 .. .. | „ „ | ♏ ½–¾. |
| 1 year .. .. | Tinct. opii (*a*)<br>*or*<br>Inj. morph. | ♏ ii.–iii.<br>gr. 1/75. |
| Over 1 year .. | Tinct. opii<br>*or*<br>Inj. morph. | ♏ ii. for each year and ♏ ii. in 15 min. if necessary.<br>gr. 1/75 for each year. |

II.—*In Adults*

| | | |
|---|---|---|
| 12–15 years .. | Tinct. opii<br>*and*<br>Inj. morph. | ♏ xxx.<br>gr. ¼. |
| 15–20 „ .. | Tinct. opii<br>*and*<br>Inj. morph. | ♏ xxx.<br>gr. ¼. |
| Over 20 „ .. | Tinct. opii<br>*and*<br>Inj. morph. (*b*)<br>*or*<br>Inj. morph. (*c*). | ♏ xxx.<br>gr. ¼–½.<br>gr. ½–¾. |

*Remarks.*—(*a*) Tinct. opii is more satisfactory than morphia. (*b*) For women. (*c*) For men according to stamina.
N.B.—Should slow or shallow breathing give rise to anxiety, atropine sulphate gr. 1/200–1/50 should be administered hypodermically.

reapply a new tannic acid dressing in order to get a good result.

The cleansing must start with the removal of all dead and charred tissues, the excision of all loose skin raised over blisters, and the removal of grease, especial attention being given to the edges of the damaged area. The whole area should then be gently but thoroughly cleansed with soap and water (applied with a sterile swab and *not* with a nail-brush) and finally carefully sponged over with ether, to remove all natural or applied grease, in order to get a thorough coagulation. It is obvious that this treatment cannot be carried out unless the patient is rendered insensible to the pain which such a cleansing of the damaged area must entail. We have found at St. Thomas's that it is safer and more satisfactory to procure this insensibility by the administration of large doses of opium rather than the use of general anæsthesia which tends, even with the greatest care in its administration, to be followed by pulmonary complications, usually of a mild nature, which are detrimental to the rapid recovery of the patient. Table V. indicates the dosage and drug employed at various ages, and has proved both safe and satisfactory.

### APPLICATION OF COMPRESS

A compress consisting of either six layers of sterile gauze or three layers of lint should be thoroughly soaked in a stock solution of 2 per cent. tannic acid and 1/2000 perchloride of mercury, and applied closely and evenly over the entire burnt area without wringing the dressing out. This compress should then be firmly and evenly bandaged in position, the bandage being applied directly to the outer side of the compress. The whole of the outside of the bandage may then be soaked with a spray of 2 per cent. tannic acid and 1/2000 perchloride of mercury in order to ensure that the underlying dressing is thoroughly wet. Mackintosh or jaconet should be placed under the patient until the dressing has dried in order to save soiling of the sheets by the tannic acid solution, for though this all washes out excepting for a faint iron mould, the sheet subsequently rots in this area. It is also well to secure the burnt limb temporarily in order to prevent undue movement until the tannic coagulum has formed firmly. This dressing should be left in position and untouched for at least a fortnight in the case of small burns and scalds, and three weeks where large areas are involved. At the end of this time the bandage should be cut and the dressing lifted, when the scab usually will separate completely from the burnt area. If it does not do so the dressing can be re-bandaged and removed later when separation has occurred. The separation may be rendered quite painless and assisted by spraying with tannic acid solution between the coagulum and the skin during this process. May I here say that the escape of a little serous or sanio-serous fluid from the edges of the coagulum or the raising in blisters of its centre with serum is no more an indication for interference or removal of the dressings during the first week after their application than it is with the bare coagulum resulting from the "spray" method. Temperature and toxæmia as shown by a rapid pulse, dried and furred tongue, and pain, with possibly the escape of sero-pus in considerable quantities, are the only indications calling for removal of these dressings before the time stated, and these indications in our experience are extremely rare and usually result from a failure in the thoroughness of the preliminary cleansing. Should removal be necessary, and in my experience it is very seldom called for, the area should be re-cleaned carefully and another tannic acid dressing applied.

As indicated, the dressing must be closely and evenly applied to the burnt area, and there are certain areas, therefore, such as the perineum, the groin, axillæ, the neck, and also the face, where the absence of bandages is an advantage, and where the spraying method gives better results, but the compress method has been and can be used with good effect in these areas. It must be realised that in burns where destruction of the subcutaneous and muscular tissues has occurred subsequent scarring and contracture are inevitable, and it is therefore necessary to apply tannic acid dressings, either by compresses or spraying, with the part in such a position that subsequent contracture may be counteracted. Equally is it obvious that although full epithelialisation will have occurred in those areas where the first, second, or third degree burn has occurred, an ulcer must be expected, and will be met with, in deeper burns, and this, if extensive, should be treated by subsequent autogenous skin-grafting, or if small by the application of some lotion, of which, in my experience, the most satisfactory is the following: purified alum, grs. 20; zinc sulphate, grs. 10; glycerin, 7 fluid ounces; water, to one pint.

Here I would like to draw attention to the excellence of this 2 per cent. tannic acid compress as a dressing not only for electrical, radium, and chemical burns, but also as an application to the area from which Thiersch grafts have been cut for the purpose of skin grafting in mastoid and other operations.

### SOLUTION

It will be seen from the above that a stock solution of 2 per cent. tannic acid and 1/2000 perchloride of mercury will fulfil adequately the needs of the compress method. In order to meet the desirability of applying this solution warm to the burnt area it is our custom to keep it in a concentrated form and dilute it with the requisite amount of warm water. Personally I do not advise keeping it at more than double strength, as I find that more concentrated solutions are inclined to decompose more quickly, and the results obtained are therefore not so painless or so satisfactory as from the weaker solution. In order to meet the requirements of shops, or works, and the home, where burns are not frequent, or on ships, where the storage of quantities of solution is likely to be a bother, I have, with the assistance of Mr. Peck, composed a tablet containing tannic acid grs. 17½, perchloride of mercury gr. ½, and boric acid (excipient) gr. 1, which tablet dissolved in 2 oz. of water gives a solution of the necessary strength though slightly muddy in appearance, due to the presence of the boric acid which, however, does not interfere in any way with its efficacy of coagulation. It can be used equally well for spraying where this method is preferred.

## Results of Tannic Acid Treatment

As will be seen from Table I., under the old bath and grease treatment, the average mortality was 39·6 per cent. burns and 18·8 per cent. scalds. In the early days of the picric acid treatment the mortality was 39·7 per cent. burns and 7 per cent. scalds, and in the last five years of the picric acid treatment the mortality was 15·5 per cent. burns and 7·5 per cent. scalds, whereas the adoption of tannic acid in October, 1928, and its systematic use in subsequent years has reduced the mortality to 4 per cent. burns and 1·7 per cent. scalds (Table IV).

If we consider the mortality in the case of children under 10 years (Table VI.), and compare them with those in the late picric acid period, it will be seen that the results with burns are not good as with scalds—a point analogous to the transition from oil to picric acid (compare Table II. and III.A). The results, however, show a distinct amelioration, and lead one to hope that further reduction in mortality may take place, and it must moreover be realised that these figures are for the severe burns alone. The Edinburgh figures (combined) for the same period are 8·7 per cent. (1926-29), 5·8 per cent. (1930), and 4·8 per cent. (1931), against St. Thomas's (combined) 3·7 per cent. (1929–32), and Sick Children's Hospital, Toronto, 16 per cent. in 230 cases (1925–28).

I would add that during the period 1929 to the present time, when tannic acid compresses have been the routine out-patient treatment for all burns and scalds, no deaths have occurred among those slighter cases which average 800 a year, of which about one-third are in children under 10 years of age. As might be expected there is a seasonal incidence of approximately 3 : 2 in favour of the winter months. For the past three years, then, the compress method

TABLE VI.—*Mortality of Burns and Scalds under 10 years in St. Thomas's Hospital*

*Treatment.*—1924–28, picric acid 2 per cent.; 1929–32, tannic acid 2 per cent. $HgCl_2$ 1/2000.

| Year. | Burns. | | | Scalds. | | |
|---|---|---|---|---|---|---|
| | No. | Deaths. | Per cent. | No. | Deaths. | Per cent. |
| 1924 .. .. | 7 | 1 | 14·2 | 23 | 0 | 0 |
| 1925 .. .. | 7 | 2 | 28·4 | 26 | 3 | 11·5 |
| 1926 .. .. | 7 | 0 | 0 | 32 | 3 | 9·3 |
| 1927 .. .. | 5 | 1 | 20·0 | 30 | 5 | 16·6 |
| 1928 .. .. | 6 | 0 | 0 | 23 | 3 | 13·0 |
| Total 5 years.. | 32 | 4 | 12·5 | 134 | 14 | 10·4 |
| 1929 .. .. | 3 | 0 | 0 | 23 | 0 | 0 |
| 1930 .. .. | 3 | 1 | 33·3 | 34 | 2 | 5·8 |
| 1931 .. .. | 14 | 1 | 7·1 | 38 | 1 | 2·6 |
| 1932 .. .. | 7 | 1 | 14·2 | 41 | 0 | 0 |
| Total 4 years.. | 27 | 3 | 11·1 | 136 | 3 | 2·2 |

has been in use in the out-patient department at St. Thomas's Hospital with excellent results. I regret I cannot give details either of the number and type of cases treated nor of the exact degree of severity of the burns and scalds; but in only two cases has sepsis occurred of a sufficiently severe nature to call for the admission of the patients to the wards, and in both these cases cleansing and reapplication of the tannic acid dressing has led to an uneventful recovery.

I venture to think that this simple method of compress may be of universal application both by the general practitioner, in the out-patient departments of hospitals, and as a first-aid treatment in works and in the home, presenting, as it does, in its simplicity great advantages over the more complicated method of reinforced compresses recently advocated by Hunt and Scott [12] and Wilson.[9]

In the foregoing remarks I have endeavoured to trace the progress of the treatment of burns and scalds during the past 40 years, and to show that although improved social conditions and an increase in the number of scalds have done much to reduce this mortality, yet improved treatment has done more, especially the introduction, or should I say rediscovery, of the value of tannic acid in this relationship. I have described a simple method of using this tannic acid, and even if you do not consider it suitable for universal application, as I hope you may, you must admit that its success in reducing the mortality of severe burns and scalds to 4 per cent. and 1·7 per cent. respectively at least redounds to the credit of the local treatment, for social conditions remain the same as in the preceding five years when the mortality was considerably higher (15·5 per cent. and 7·5 per cent.).

Further, may I urge the necessity of separating the figures relative to the treatment of burns from those of the treatment of scalds in papers dealing with this subject.

Finally, I wish to tender my thanks, in addition to those already mentioned, to my successive house surgeons, Messrs. R. W. Butler, R. O. Lee, T. H. Wilson, R. H. Franklin, and T. W. Mimpriss, and especially to Miss Routh (sister of the septic wards), to whose patient care and continued enthusiasm much of the success of this treatment has been due.

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# IDIOPATHIC APLASTIC ANÆMIA

## WITH AN ANALYSIS OF FOUR CASES

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APLASIA of the bone-marrow is a condition in which the hæmopoietic function of the marrow has become depressed, so that there is a diminution in the output of those blood-cells which are derived from it—i.e., the erythrocytes, the granulocytes, and the platelets.

It is not so much a disease-entity as a state produced by many conditions, such as poisoning by benzol and its derivatives; excessive exposure to irradiation; exhaustion of the bone-marrow by certain types of anæmia, such as hæmolytic anæmia and those anæmias with a megalocytic blood picture; and certain diseases which may affect the bone-marrow such as tumours and leukæmia. These conditions so depress the function of the bone-marrow that it ceases, to a greater or lesser extent, from performing its task of producing blood-cells. It is said that sepsis can produce aplasia. This and the pathogenesis of aplastic anæmia have been discussed elsewhere.[1]

But when these conditions of known ætiology have been excluded, there remains one type of aplasia, consistent in its symptoms, blood picture, and clinical history, and of no known causation, which may be regarded as a disease entity, and to which the term

idiopathic aplastic anæmia is applied. It is this condition that is considered in this paper.

The symptoms and physical signs are dependent on the altered condition of the blood and they are, most commonly, pallor and shortness of breath, hæmorrhages into the skin or from the mucous membranes, and sepsis, generally of the fauces, which occurs late in the course of the disease.

This disease occurs particularly in young adults, but no age is exempt, and females are said to be more susceptible than males. It usually runs an acute course, with a fatal termination in a few weeks, but rare cases have been described in this country and in America in which the disease runs a more chronic course, and the patient is kept in reasonable health for a time by repeated blood transfusions. This type would be more accurately termed chronic hypoplastic anæmia, and the first of our cases described below belongs to it.

### BLOOD EXAMINATION

The characteristic feature of the blood picture in aplastic anæmia is the great reduction of all the cells derived from the bone-marrow, together with absence of or very little evidence of erythrocytic regeneration. Dyke [2] says that the detection of a single embryonic cell in a blood film is sufficient to set aside aplastic anæmia as a diagnosis. This is not quite in accord with the experience of other observers, and certainly not with our own, but this statement emphasises the rarity of primitive red or white cells in the blood in this condition.

Although the erythrocytes may number a million or less, there is no anisocytosis and no poikilocytosis. The cells are well filled with hæmoglobin and the colour-index is generally round about or above unity.

Nucleated red cells are very rarely present, and the reticulocyte count is low, usually less than 1 per cent. Minot [3] has pointed out that transient increases even above the normal percentage may occur, and in Witts's [4] case the reticulocyte count rose on one occasion as high as 6 per cent. This is clear evidence that some normal or even hyperplastic marrow is still functioning.

The great reduction of the erythrocyte count is not due to any hæmolysis, for this is not found in aplastic anæmia, but to deficient production by the bone-marrow.

There is a marked leucopenia, but only affecting the polymorphonuclear and eosinophil cells; the lymphocytes are unaffected, and the lymphocytosis is apparent and not absolute. This reduction of granulocytes is sometimes so extreme that, late in the disease, it is impossible to detect even one polymorphonuclear cell in a blood film; if these cells are reduced below 1000 per c.mm. the resistance to infection is impaired and sepsis is likely to result.

The blood-platelets are reduced, but purpura does not result until their number is greatly diminished, usually well below 60,000 per c.mm. This marked thrombocytopenia is often seen late in the course of the disease. Whenever purpura is present, the bleeding time is prolonged and the capillary-resistance test is positive. Many authorities consider that the coagulation time is lengthened, but Minot [3] finds it normal or only slightly delayed. This author says that the fragility of the erythrocytes is abnormal, the cells beginning to break up in about the normal strength of salt solution, but that complete hæmolysis is found in a stronger solution than normal. Only in one of our cases was this test done, and in her the fragility proved to be within normal limits as tested against a control.

### PATHOLOGY

The morbid anatomy of the disease is illustrated by Cases 2, 3, and 4 (vide infra). The most important negative finding is the absence of hæmosiderin in the tissues; in the occasional cases where this pigment has been reported as present, it must be attributed to hæmorrhages which have taken place, since there is no hæmolysis in idiopathic aplastic anæmia.

The positive finding of importance is the characteristic appearance of the bone-marrow, best described by Sheard.[5] The marrow cavities of the long bones are filled with yellowish-white fat, sometimes of the consistency of pus. Microscopically, all the bone-marrow cells are diminished or absent. As a rule, this fatty change is not complete, and there remain, even in the long bones, a few small scattered areas of active marrow; while it is almost customary to find that the marrow of the cancellous bones—e.g., the sternum and ribs—remains active. Owing to the defective blood-supply various internal organs will be affected with fatty degeneration.

### DIFFERENTIAL DIAGNOSIS

*From pernicious anæmia.*—Aplastic anæmia is a disease entirely distinct in its ætiology, pathology, and symptoms from pernicious anæmia, and it is important that these two conditions should be differentiated, since the prognosis and treatment are so different. The chief points of distinction are:—

(1) The average age-incidence is higher in pernicious anæmia.

(2) Glossitis, subacute combined degeneration of the cord, and splenomegaly may be present in pernicious anæmia; these conditions do not occur in aplastic anæmia.

(3) Achlorhydria is practically always present in pernicious anæmia; this is not so in aplastic anæmia.

(4) Clinical and pathological evidence of hæmolysis is present in pernicious anæmia and absent in aplastic anæmia.

(5) The average size of the red cell is increased in pernicious anæmia and normal in aplastic anæmia.

(6) Evidence of attempted regeneration of the erythrocytes is always obtained in pernicious anæmia and is typically absent in aplastic anæmia.

(7) Aplastic anæmia does not respond when the missing hæmatinic factor of pernicious anæmia is supplied.

The distinction is not, however, always so simple as these categorical statements suggest, for it is possible to get an aplastic blood picture in pernicious anæmia when the disease is rapidly progressive; when sepsis is present; or when the disease is not under suitable treatment—i.e., when the dose of the hæmatinic factor is insufficient or the preparation used is inactive. The blood picture of these cases shows, in addition to a marked diminution in the number of the erythrocytes, granulocytes, and platelets, no sign of red cell regeneration.

The following case illustrates the difficulty when an aplastic blood picture is present in pernicious anæmia.

A man, aged 56, had suffered in February, 1931, from a severe attack of obstructive jaundice, which cleared up in a few weeks. About a month later it was noticed that he had become very pale. His blood count gave 780,000 red cells, 2400 white cells, 26 per cent. of hæmoglobin, and a colour-index of 1·6. Examination of a blood film showed no anisocytosis or polychromasia, and no nucleated red cells. Nevertheless he improved with a blood transfusion and regular doses of ventriculin, and in two months he was in good health and back at his work, taking a small dose of liver extract each day.

At autopsy an examination of the marrow in the two diseases is distinctive, though Witts [6] says that if a thorough examination of the marrow is made in any case of pernicious anæmia, areas of complete aplasia are always found. Minot [7] refers to cases in which the marrow is hyperplastic yet the blood examinations have shown evidence of aplasia. In other megaloblastic anæmias which may appear in

the course of certain diseases with gastro-intestinal lesions, such as sprue, fatty and chylous diarrhœa, cœliac disease, pellagra, and infestation by certain parasites, the marrow can become exhausted and a blood picture of aplastic anæmia may result.[6]

*From idiopathic purpura hæmorrhagica and agranulocytic angina.*—Aplastic anæmia can usually be differentiated clinically from these two diseases. In purpura hæmorrhagica the hæmorrhages and purpura occur early, before the anæmia is evident. The anæmia is secondary to the blood loss, and signs of red cell regeneration are usually obvious in an examination of a blood film. (This is of great practical importance if the question of splenectomy arises, as one of the contra-indications is a degree of anæmia greater than would be accounted for by the hæmorrhage.) Thrombocytopenia is present in both conditions.

In agranulocytic angina signs of infection, generally in the fauces, accompanied by a profound granulopenia, occur at the onset. Characteristically there is no anæmia, and the platelets are normal in number. The diagnosis is not always easy, however; it is customary in aplastic anæmia for the hæmopoietic function of the marrow to be affected in this order: first the red cells, then the granulocytes, and lastly the platelets; but the order may be reversed, so that if the platelets fail early purpura hæmorrhagica will be simulated; but if the leucocytes are affected early, the clinical picture may resemble that of agranulocytic angina. In the second and third of our cases (vide infra) these particular difficulties in diagnosis were encountered.

Moreover, there are cases which are definitely intermediate between these three conditions, and in these it may be impossible to make a clear-cut diagnosis. Tidy[8] has drawn attention to cases of purpura hæmorrhagica in which there is evidence of some degree of aplasia of the bone-marrow with an almost complete absence of neutrophils. Minot[3] says:—

> "There are many cases in the literature which are intermediate between idiopathic aplastic anæmia with symptomatic purpura hæmorrhagica, and idiopathic purpura hæmorrhagica itself. From such cases it is suggested that these two conditions may perhaps be not distantly related, for one can find cases of these idiopathic conditions, where the blood picture can be interpreted as showing all degrees of involvement of the three chief elements of the bone-marrow."

We have suggested elsewhere[1] that these three conditions should not be regarded so much as clear-cut entities, but rather as differing stages of one disease whose cause is a primary deficiency of the bone-marrow.

*From aleukæmic leukæmia.*—This is a variety of leukæmia which may run part or the whole of its course without any increase in the number of leucocytes circulating in the blood stream. Aplastic anæmia is thus simulated, since the blood picture may show an anæmia, a thrombocytopenia, and a leucopenia with a relative or an absolute lymphocytosis. Clinically, fever, sepsis, and hæmorrhages may be present without obvious enlargement of the spleen and lymphatic glands. A critical survey of the blood picture should show, at some phase of the illness, a lymphocytosis, perhaps up to 90 per cent.; signs of red cell regeneration; and, even more important, lymphoblastic cells. Nevertheless, it is sometimes impossible to make a diagnosis until autopsy when, instead of the acellular marrow of aplastic anæmia, an enormous lymphocytic hyperplasia is found.

*From hæmolytic anæmia.*—This is a rare disease which is not likely to be mistaken for aplastic anæmia. The evidence of increased destruction of red cells in the circulating blood and an attempt at an increased formation of these cells by the bone-marrow is usually obtained without difficulty. The two clinical signs, icterus and splenomegaly, are sufficient to negative a diagnosis of aplastic anæmia, even though an aplastic blood picture develops, as it sometimes does, as a terminal event.

*From severe anæmia which does not conform to any particular group.*—This type of anæmia is seen more particularly in infancy. Witts[9] says that there are plastic and aplastic groups with intermediate forms of a hypoplastic type. Death may result from the complete failure of the marrow or, more often than in adults, there is a gradual resumption of function. A case reported by Hutchison[10] seems to belong to this group.

## PROGNOSIS

In idiopathic aplastic anæmia the outlook depends on the degree of involvement of the bone-marrow; when much of the bone-marrow is affected, as is usually the case, the prognosis is almost hopeless, but in the rare cases of chronic hypoplastic anæmia some good may result from treatment. We have found only six reported cases of aplastic anæmia which have benefited materially by treatment; of these, two have since relapsed and died.

Stewart Harrison[11] and Parkes Weber[12] have each reported a case of chronic hypoplastic anæmia. In the former case the diagnosis was made as far back as 1925 and the patient has been treated with blood transfusions, repeated every few weeks; at present he is able to do his work and maintain moderate health. Hurst, in a personal communication five months ago, wrote that this man remained well and had just received his 144th blood transfusion. Weber's case dated from January, 1931; at the date of reporting he has had 16 blood transfusions and remained fairly well. This case proved later, at autopsy, to be one of aleukæmic myelosis (*Quart. Jour. Med.*, 1932, xxv., 409).

Herman's[13] patient was a child whose blood suggested aplastic anæmia and who improved after one blood transfusion. She died, however, of anæmia four years later, and no autopsy was obtained.

Gibson's very interesting case[14, 15] was a girl, aged 11, who suffered from aplastic anæmia of an advanced type but was kept in fair health for over six years by daily injections of adrenaline. Her condition had not been improved by blood transfusion. She died of hæmorrhagic chicken-pox.

Parkinson[16] and Hayes Smith[17] have each reported a case. In both patients purpuric manifestations appeared at the onset of the illness, to be followed by anæmia. A blood film of Parkinson's patient showed some signs of erythrocytic regeneration. For these reasons it is possible that they were examples of purpura hæmorrhagica, with some temporary aplasia of the bone-marrow.

Alpham and Nelson[18] have reported a man suffering from severe aplastic anæmia, who showed no improvement after blood transfusions and treatment with calf's liver. With massive doses (1000 g. per day) of fœtal calf's liver he recovered, and kept well on a much smaller dose.

These few recoveries and remissions do little to lighten an almost hopeless prognosis.

Lately, at Prof. Tanhauser's clinic at Freiburg, a young woman was seen who developed in the later weeks of pregnancy a severe anæmia with an aplastic-like blood picture. Intensive liver therapy and repeated blood transfusions improved her condition but little. Later, after many septic teeth had been removed and without further treatment, a rapid improvement set in. In this case a diagnosis of hæmolytic anæmia was definitely ruled out, and Tanhauser's opinion was that she was a type of aplastic anæmia. Whether the removal of septic teeth contributed to the improvement it is difficult to say. Witts,[18] in his recent Goulstonian lectures, quoted Larrabee's remark that "a man's index of scepticism must indeed be low if he can believe that these terrible,

acute anæmias are the result of chronic foci of infection in teeth or tonsils, and can bring himself to hope for relief from their eradication." However this may be, such cases as that quoted from Freiburg do occasionally occur. They are a reminder that, although enormous advance has been made in the last ten years in the diagnosis and classification of anæmias, clinical difficulties have not yet disappeared. It is still wise to be guarded in prognosis until the diagnosis is securely established.

### TREATMENT

The success of treatment in idiopathic aplastic anæmia will depend on the degree of deficiency of the circulating blood elements, which in turn will depend on the amount of bone-marrow which remains plastic, and which is capable of stimulation. The bone-marrow stimulants which are at present known include blood transfusions, small doses of X rays applied to the long bones, foreign proteins (such as milk), and certain endocrine extracts—e.g., adrenaline. Iron, arsenic, liver therapy, and removal of septic foci are considered to be unavailing.

### DESCRIPTION OF CASES

The following four cases of aplastic anæmia have been under the care of one of us in the Derbyshire Royal Infirmary.

#### THE FIRST CASE

A woman, aged 32, was admitted to hospital for the first time in November, 1931, with a history that she had always been healthy until about two months before, when she had noticed that she was losing energy and was becoming weak and short of breath. She had had three children; their births had been attended with no difficulty, nor had there been any great loss of blood. The periods had always been quite regular and the loss not excessive.

She was a well-nourished though very anæmic woman, but in spite of this she said that she felt fairly well. The skin and conjunctiva were pale, but there was no lemon colour of the skin and no icteric tinge of the conjunctiva. There were several infected teeth, and the tonsils were enlarged and showed some signs of chronic inflammation, though the tonsillar glands were not affected. No further sign of sepsis was found. There was no increase of temperature. No abnormality was found in the tongue, lungs, or abdominal, urinary, and endocrine organs, nor in the nervous system (with the exception of two small hæmorrhages in the left retina). Neither the spleen nor any of the lymphatic glands were felt to be enlarged. There were a few petechiæ scattered over the trunk and thighs. There was no evidence of any blood destruction. There was some dilatation of the heart, as shown by the position of the apex beat, which was 3¼ in. from the midsternal line, with a softening of the first sound.

The result of the blood examinations on various dates are shown in Table I. In addition, the red cells were found always to be of normal shape and size, and no abnormal cells or reticulocytes were seen. The bleeding time was 5½ minutes, and the coagulation time 2¼ minutes (Brodie Russell). The fragility of the red cells appeared to be normal.

| | Patient. | | Control. |
|---|---|---|---|
| Hæmolysis started .. | 0·45 % saline | .... | 0·45 % saline |
| „ completed.. | 0·35 % „ | .... | 0·35 % „ |

Van den Bergh's reaction was completely negative, both directly and indirectly. A fractional test-meal showed free

TABLE I.—*Blood Examination (Case 1)*

| Date. | Remarks. | Hb. per cent. | Red cells per c.mm. | Colour-index. | White cells per c.mm. | Differential count per cent. | | Size of red cells. | Platelets per c.mm. | Reticulocytes per cent. |
|---|---|---|---|---|---|---|---|---|---|---|
| | | | | | | Pms. | Lms. | | | |
| 26/11/31 .. | — | 40 | 1,310,000 | 1·5 | 3,200 | 36 | 63 | 7·17 | 30,000 | 0 |
| 27/11/31 .. | Liver, ventriculin, iron started. | — | — | — | — | — | — | — | — | — |
| 10/12/31 .. | — | 30 | 1,070,000 | 1·4 | 4,300 | — | — | — | — | — |
| 14/12/31 .. | — | 30 | 1,080,000 | 1·4 | — | — | — | — | — | — |
| 30/12/31 .. | — | 20 | 790,000 | 1·4 | 3,100 | — | — | — | — | 1 |
| 30/12/31 .. | Injections of adrenaline started. | — | — | — | — | — | — | — | — | — |
| 5/1/32 .. | — | 30 | 940,000 | 1·5 | 4,800 | 28 | 71 | 7·45 | 94,000 | 1 |
| 6/1/32 .. | B.T., 1 pint. | — | — | — | — | — | — | — | — | — |
| 8/1/32 .. | — | 40 | 1,320,000 | 1·5 | 1,600 | 24 | 74 | 6·74 | — | — |
| 14/1/32 .. | — | 35 | 1,590,000 | 1·1 | 3,200 | — | — | — | — | — |
| 20/1/32 .. | — | 25 | 1,300,000 | 1·1 | 1,600 | 24 | 72 | 7·64 | 73,000 | — |
| 28/1/32 .. | — | 30 | 1,090,000 | 1·5 | 2,400 | 16 | 81 | 7·17 | — | — |
| 1/2/32 .. | B.T., 1 pint. Adrenaline stopped. | — | — | — | — | — | — | — | — | — |
| 9/2/32 .. | — | 40 | 1,660,000 | 1·5 | 4,000 | 31 | 64 | 7·20 | — | — |
| 21/3/32 .. | — | 10 | 790,000 | — | 2,400 | — | — | — | — | — |
| 22/3/32 .. | B.T., 1 pint. Adrenaline injec. restarted. | — | — | — | — | — | — | — | — | — |
| 24/3/32 .. | B.T., 1 pint. | — | — | — | — | — | — | — | — | — |
| 26/3/32 .. | „ | — | — | — | — | — | — | — | — | — |
| 31/3/32 .. | — | 60 | 2,240,000 | — | 4,000 | 38 | 58 | — | — | — |
| 4/5/32 .. | — | 20 | 820,000 | — | 2,400 | — | — | — | 20,000 | 2 |
| 5/5/32 .. | B.T., 1 pint. | — | — | — | — | — | — | — | — | — |
| 8/5/32 .. | — | 40 | 1,960,000 | 1·0 | 4,000 | — | — | — | — | 1 |
| 1/6/32 .. | B.T., 1 pint. | — | — | — | — | — | — | — | — | — |
| 7/6/32 .. | — | 20 | 1,800,000 | 1·1 | 3,200 | 23 | 71 | — | — | 1 |
| 23/6/32 .. | — | 15 | 600,000 | 1·2 | 2,800 | — | — | — | 7,000 | — |
| 24/6/32 .. | B.T., 1 pint. | — | — | — | — | — | — | — | — | — |
| 25/6/32 .. | — | 35 | 1,600,000 | 1·1 | 3,000 | — | — | — | — | — |

Pms.= Polymorphonuclears; Lms.= Lymphocytes. B.T.= Blood transfusion.

acid in normal amounts. A series of radiograms taken at intervals after a barium meal showed no abnormal shadows in the stomach or intestines. The stools appeared normal and no ova were seen in them. One of the infected teeth was removed, but no streptococci were found on culture from it.

During the first month in hospital she was treated with iron in large doses, ventriculin, and a liver preparation both by the mouth and intramuscularly (5 c.cm. of Hepatex P.A.F. daily). As was to be expected, there was no improvement either in her general condition or in her blood, as shown by absence of increase in the hæmoglobin, erythrocytes, or reticulocytes. On Dec. 30th, after we had read an account of Gibson's successful treatment of a girl suffering from this disease, an injection of 3 minims of adrenaline 1/1000 was given, and this was continued daily for nearly a month. In six days there was an improvement in her general condition, and to some extent in her blood.

On Jan. 6th, 1932, she was transfused with a pint of citrated blood, with great improvement. This was repeated on Feb. 1st and the improvement was maintained until about the middle of the month, when she began to relapse. So, in an effort to stimulate the marrow as much as possible, three large transfusions were given at intervals of two days, and the adrenaline injections resumed daily. She was able to go home and even to do part of her daily work, but by the beginning of May more transfusions became necessary. She improved again, and arrangements were made for her to have one or two blood transfusions every month. Since then, however, the improvement has not been maintained for as long as formerly, and a transfusion is necessary about every two to three weeks. The benefit from these transfusions only lasts about two weeks, after which she quickly

becomes anæmic, with extensive purpuric hæmorrhages. This patient died in July, 1932; unfortunately, no autopsy, was obtainable, so that the diagnosis in this case cannot be regarded as definitely established.

THE SECOND CASE

A boy, aged 13, was admitted into hospital on Nov. 26th, 1931, suffering from extreme anæmia. He had always been well until the last five weeks, when he had become pale, with nose-bleeding for one day just before admission. He was extremely ill, and his pallor was intense. There was no icteric staining of the conjunctivæ or skin. There were a number of petechial hæmorrhages scattered over the trunk and limbs, with some bruising of the soft tissues. A systolic murmur was heard all over the præcordium. The liver and spleen were not felt. There were no retinal hæmorrhages. The temperature was 97° F., but rose, and varied between 100° F. and 103° F. The pulse-rate kept above 140 per minute. There was no sign of sepsis. A blood examination gave the following results: 400,000 red cells, 10 per cent. hæmoglobin, a colour-index of 1·2, and 2000 white cells. The average size of the red cell was 7·48 μ (by Eve's halometer). A differential count showed polymorphonuclear cells 41 per cent. and lymphocytes 59 per cent. The erythrocytes were of normal shape but irregular in size, and a few normoblasts and megaloblasts were seen. The blood-platelets numbered 10,000 per c.mm. The bleeding time was 13 minutes, and the coagulation time 24 minutes (Brodie-Russell). With a van den Bergh's test there was a faint positive indirect reaction. The tourniquet test was definitely positive.

Soon after admission the boy was given a transfusion of 350 c.cm. of citrated blood. An injection of 3 minims of 1/1000 solution of adrenaline was ordered every four hours, and hepatex P.A.F. 5 c.cm. was injected intramuscularly once a day. On the next day the boy was decidedly better, but he collapsed the day after and died.

At the *post-mortem examination* the left femur was split open longitudinally, so as to expose the marrow of the diaphysis and epiphysis. The marrow was of a yellow-white colour, about the consistency of thick pus, with a few small scattered pink areas. That of the sternum and several of the ribs was normal in appearance.

On *microscopical examination* of the marrow from the femur no cells were seen, only fat. From that of the rib, however, red cells, both nucleated and non-nucleated, were seen, with a few granulocytes and myelocytes; no organisms were found in the marrow. There were a number of petechial hæmorrhages on the visceral layer of the pericardium. The heart was dilated. There was evidence of some bronchopneumonia (terminal) of the right lung. The liver and spleen (3 oz.) were not enlarged, and looked normal. Some of the mesenteric glands were caseous. There was a persistent thymus, but the other endocrine glands were normal macroscopically.

THE THIRD CASE

The third patient was a man aged 40, a sorter, who had always been healthy except for malaria contracted during the war, and piles which bled from time to time. About the middle of April, 1931, he began to complain of giddiness and distaste for food, with abdominal discomfort. Some degree of jaundice and pyrexia was noticed. After about ten days' treatment he improved and went back to work, only to return to his bed a few days later as the weakness had increased. On admission to hospital on May 16th he looked very ill and pale, and was in a typhoid state. The tonsils were covered with an extensive slough which had spread on to the soft palate. There was no glossitis, nor was there any sign of hæmolysis of the blood or of jaundice. Nothing abnormal was found in the chest, in the abdomen, or in the nervous system. The urine contained a trace of albumin. The temperature ranged between 99° F. and 103° F. There were no hæmorrhages into the skin or retinæ, or from the mucous membranes. A blood count on the day of admission showed 25 per cent. of hæmoglobin; 1,840,000 red cells per c.mm., and a colour-index of 0·9. The size of the blood corpuscles was 7·46 μ. There were 400 white cells per c.mm., but no granulocytes were seen; the lymphocytes numbered 88 per cent., and the large monocytes 12 per cent. No signs of attempt at red cell regeneration were to be found.

He died a short time after admission to hospital. On *autopsy* the marrow from the femora was pale and fatty; on microscopical examination no normoblasts, granulocytes, or myelocytes were to be seen, the marrow being composed entirely of fat. The marrow from the sternum and ribs, however, was normal.

THE FOURTH CASE

The patient, a man aged 44, was a clerk employed in a factory where benzol compounds were used; he was seen on Jan. 10th, 1928. His work, he said, confined him to the office only, and he had never come in contact with the chemicals. He had been quite well until about six weeks before, when he noticed that he was becoming weak and breathless. He had been told that he was becoming pale.

On examination he looked a well-nourished man, but showed marked pallor. There were no enlarged glands, and the spleen and liver appeared normal. Nothing wrong was detected in his chest or abdomen except some dilatation of his heart. There was no sign of sepsis. He had some oozing of blood from his gums, but there was no clinical sign of blood destruction.

TABLE II

| Date. | Hb. per cent. | Red cells per c.mm. | White cells per c.mm. | Granulocytes per cent. | Lymphocytes per cent. |
|---|---|---|---|---|---|
| Jan. 10th | 35 | 1,500,000 | 4,400 | 11 | 89 |
| „ 11th | 30 | 1,090,560 | 1,600 | 41 | 58 |
| „ 12th | 20 | 860,000 | 400 | 8 | 92 |

A series of blood examinations is shown in Table II. There was no sign of any attempt at erythrocytic regeneration. On *autopsy* the marrow of both femora was examined, and practically all the hæmopoietic tissue was found to be replaced by fat. Microscopically no cells were seen except a few mononuclear ones.

COMMENTARY

The first of these cases is the most interesting, and undoubtedly belongs to the group named chronic hypoplastic anæmia, of which very few examples have been reported. The possible alternative diagnoses are pernicious anæmia with an aplastic blood picture, and lymphatic leukæmia in an aleukæmic phase. The diagnosis of pernicious anæmia is easily dismissed by the absence of the necessary clinical and pathological criteria, and by the steady clinical and hæmatological deterioration under intensive liver therapy.

The diagnosis of aleukæmic leukæmia deserves more serious consideration, for in a few of the differential white cell counts there is a slight but absolute lymphocytosis; in one instance only, however, did these cells number over 3000 per c.mm., and that was a few days after injections of adrenaline had been started (adrenaline produces a transient lymphocytosis). Against the diagnosis of leukæmia is the absence of embryonic types of lymphocyte and of any sign of red cell regeneration; and the fact that, although the case has been under observation for some months, no enlarged lymphatic glands have been detected. Although this diagnosis cannot be dogmatically dismissed, yet the patient's reaction to blood transfusions and adrenaline injections suggest that the diagnosis of chronic hypoplastic anæmia is likely to be the correct one. An interesting observation in this case was that there were no purpuric manifestations until the platelet count had fallen to 30,000 per c.mm.

In the second case the blood loss was not sufficient to cause the grossly anæmic blood picture, and the fact that the child had been ill and noticeably anæmic for many days before hæmorrhages or purpura were observed establishes the differential diagnosis from purpura hæmorrhagica. There were a few signs of attempted blood regeneration, but it should be remembered that in children the bone-marrow is more labile than in adults; moreover, some signs of red cell regeneration do not rule out aplastic anæmia. Cabot[2] has reported a man suffering from aplastic anæmia, whose blood film showed marked evidence of regeneration, but on autopsy typical aplasia of the marrow was seen. Acute leukæmia was a possible diagnosis during life, but this was definitely excluded by the post-mortem examination. There was no clinical sign of hæmolysis, and no hæmosiderosis was seen at autopsy, yet the van den Bergh's test showed a slight positive

indirect reaction (probably this is without diagnostic value), so that it is possible, but unlikely, that this was an example of the acute hæmolytic anæmia in children which terminates in aplasia, and which has been described by Kleinschmidt (quoted by Minot [3]).

In Case 3 the gross leucopenia with complete absence of granulocytes, and the predominant picture of infection and fever, suggest a diagnosis of agranulocytic angina; but the severe anæmia and the aplasia of the bone-marrow at autopsy place it in an intermediate group between aplastic anæmia and agranulocytic angina. A platelet count might have confirmed this, but unfortunately it was not done; it is significant, however, that this was the only case in our series to be without purpura or hæmorrhages, and it was also the only case to show evidence of sepsis; but in others there was not such an extreme granulopenia, except terminally in Case 4.

The morbid anatomy of the bone-marrow in the cases that came to autopsy conformed to the classical descriptions.[5] In the two adults the whole of the femoral marrow showed the typical yellow appearance of complete aplasia, but in the child there were a few isolated pink areas with a small cell content. The marrow from the sternum in all three cases appeared normal both to the naked eye and microscopically. Dyke [21] has reported various kinds of micro-organisms in the bone-marrow in cases of aplastic anæmia, but careful search failed to reveal them in our cases.

Our thanks are due to Dr. Hugh Barber for his kindly help and criticism in the preparation of this paper, and to the staff of the pathological department of the Derbyshire Royal Infirmary for undertaking all the blood examinations.

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DONATIONS AND BEQUESTS.—The late Mr. Charles Wilson, of Belsize Park-gardens, London, left £500 to the Paddington Green Children's Hospital, and the residue of the property after other bequests between St. Thomas's Hospital, London Hospital, Charing Cross Hospital, the Hospital for Sick Children, Great Ormond-street, the Royal Free Hospital, the Cancer Hospital, Fulham-road, the London Fever Hospital, the Temperance Hospital, Hampstead-road, and the Hampstead General Hospital. The value of the estate is over £200,000.—Mrs. Martha Jones, of Edgbaston, left by will £500 to the General Hospital, Birmingham; £500 to Queen's Hospital, Birmingham; £500 to the Children's Hospital, Birmingham (King Edward VII. Memorial); £500 to the Birmingham and Midland Eye Hospital; £100 to the Midland Hospital for Homœopathic and General Treatment, Birmingham; £100 to the Birmingham and Midland Hospital for Women; £100 to the Birmingham and Midland Skin and Urinary Hospital; and £100 to Dr. Barnardo's Homes.—By the will of the late Mr. William Lyle, of Welwyn, Herts, the Hertford Cottage Hospital will receive £5000, the Hertford and District Nursing Association £1000, and the Welwyn Cottage Hospital £500.

# A NEW FORM OF BED TENT FOR ADMINISTERING OXYGEN AND CARBON DIOXIDE

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*With an Account of a*
CLINICAL OXYGEN-MEASURING PIPETTE, *by*
E. P. POULTON *and* J. W. SHACKLE, M.R.C.S. ENG.

MANY methods have been employed for administering oxygen, but none of them is altogether satisfactory.

The *nasal catheter* has the advantage of simplicity, and it does not unduly inconvenience the patient; it was used first by the late Prof. Adrian Stokes of Guy's Hospital during the war, and it is the method most widely employed to-day. The proportion of oxygen in the alveolar air is raised to 24 per cent. with the oxygen flowing at 2 litres a minute; this means a brisk turbulent flow in the bottle through which the gas is usually bubbled; 24 per cent. is less than twice the normal value, so that the method cannot be regarded as really effective.

Higher values for the alveolar oxygen may be obtained by using a *face mask* as in Haldane's apparatus, or the various modifications described by H. W. Davies and A. R. Gilchrist,[1] and by R. Hilton [2]; but breathless patients are apt to resent all these devices, and the face mask is usually refused altogether. Hence it is not surprising that other more tolerable methods of giving oxygen have been elaborated, such as the oxygen chamber and the bed tent provide.

The discredited *glass funnel* that is still often supplied by chemists when oxygen is ordered was found by Hilton to cause no increase whatever in the alveolar oxygen at any rate of flow when held 10 cm. (4 in.) in front of the mouth; but when held so as to touch the nose and chin it has, with an oxygen flow of 2 litres a minute, a rather smaller effect than the nasal catheter.

Whenever oxygen therapy is discussed the question of *subcutaneous oxygen* is usually raised. If the results are beneficial they may be due to counter-irritation or acupuncture; but they certainly cannot be due to the oxygen acting as a respiratory gas, for Davies and Rabinovich [3] produced anoxæmia in dogs from pulmonary embolism; the oxygen saturation of the arterial blood was made normal by giving oxygen through the lungs; but there was no effect with subcutaneous and intraperitoneal oxygen.

## HISTORY OF OXYGEN CHAMBERS AND TENTS

The first oxygen chamber was used at Cambridge during and after the war, and results with patients were published by Barcroft, Hunt, and Dufton.[4] There are oxygen chambers at several clinics in America, and A. L. Barach has devised a portable chamber. The experiences with the oxygen chamber which was in use for some years at Guy's Hospital were briefly described by Campbell and myself.[5] It was a large chamber with three beds, and it required 600 cubic feet of oxygen to begin with, and later from 300 to 600 cubic feet every 12 hours, as well as alkali to absorb the $CO_2$.

It is clear that the stationary oxygen chamber will never be of general clinical application, and the oxygen bed tent has most of its virtues and none of its failings. The object of the tent is to enable the

patient to stay any length of time in an atmosphere enriched to any desired extent with oxygen, and containing $CO_2$ as well if required; the heat generated by the patient must be removed, and also the moisture from his breath amounting to as much as 10 oz. in the 24 hours.

The first bed tent was devised by Sir Leonard Hill.[6] Its disadvantage was that there was no adequate means of keeping the patient cool; but it was provided with a motor fan in an outer circuit to draw the air from the tent and pass it through a tin of soda lime and back again to the tent. The next advance was made by Barach,[7] who added a box to the circuit large enough to hold over 100 lb. of ice, and this has to be replenished every 12 hours; the shape of tent and "sleeve" described by him are now generally adopted. The next great advance was made by Cecil and Plummer [8] who introduced the injector, which replaced the motor fan in the circuit. In the injector the oxygen comes out under pressure through a minute orifice into the circuit, and the energy liberated is used to draw air round the circuit, so that the ventilation is effected by the potential energy of the oxygen stored in the cylinder; thus the working of the tent is independent of an electrical supply.

### THE AUTHOR'S TENT *

In my experience the greatest difficulty in treating a patient by means of an oxygen tent is to keep him dry and cool. In the plan adopted by Barach and by Cecil and Plummer a high rate of ventilation is required for this purpose, though a much lower rate is sufficient to remove the $CO_2$. In the Cecil-Plummer apparatus a ventilation of 175 litres of air is used, and this requires an oxygen feed of 5 litres a minute, which produces an atmosphere containing about 50 per cent. of oxygen, and yet if the tent is reasonably air-tight I have found that from 1 to 2 litres of oxygen a minute is enough to keep the oxygen up to this amount. By keeping the mechanism for cooling and drying separate from the $CO_2$ absorption circuit a great saving of oxygen is effected.

* The tent is being marketed by Messrs. Siebe Gorman and Co., Ltd., 187, Westminster Bridge-road, London, S.E. 1. Inquiries from the midlands should be made to Mr. J. Muirhead, 11, Cannon Hill-road, Birmingham.

FIG. 1

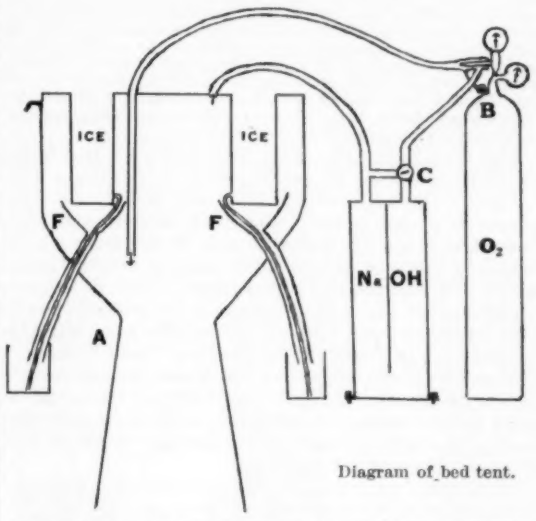
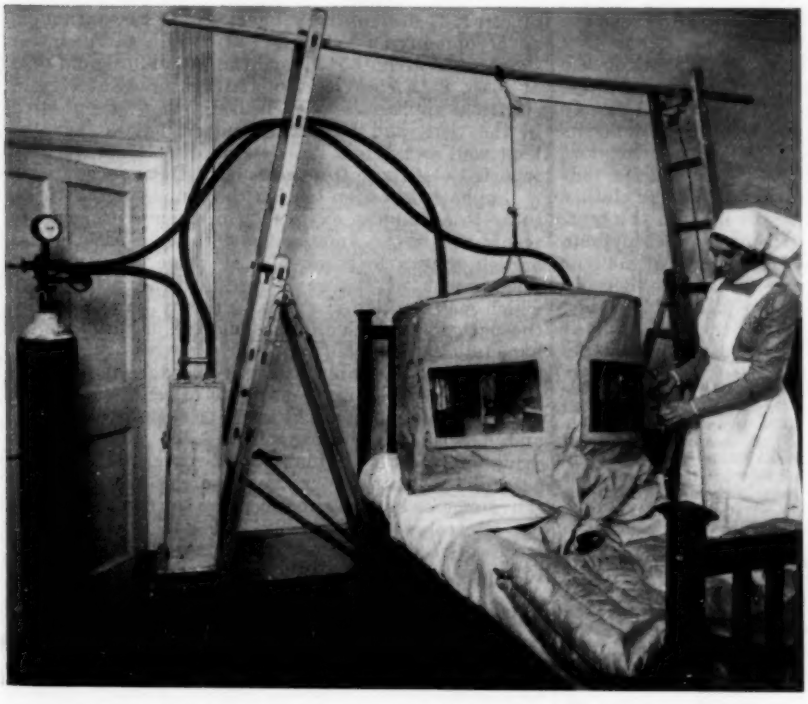



Diagram of bed tent.

The mechanism consists of two tins with radiating fins which are let in through the roof of the tent and fixed in position by bolts which can be removed at will. The tins are filled with lumps of ice or ice and salt in layers, so that the air inside the tent is cooled, and the moisture from the patient condenses on the surface and on the fins of the two tins, and runs off down the funnels (Fig. 1, F) to be collected outside the tent. As the ice melts the water can be run off by means of a thin rubber tube, which is threaded through the wider tube from the funnels. As a mixture of ice and water is of the same temperature as melting ice it is an obvious economy of ice to wait until most of it is melted before running off the water. As little as 36 lb. of ice has been required in the 24 hours, which costs less than 1*s.* 6*d.* at wholesale prices. Both tubes can be controlled by a single clip outside the tent; but as the inner tube from the ice tin is longer than the outer tube the flow of water from the ice tin can be controlled separately by a clip, and in this case it is essential that the end of the wide tube should lie beneath water, or otherwise there will be a leak from the tent through the funnel to the outside air. The funnels are made of the same waterproof fabric as the tent, and are suspended from the roof by means of fine chains.

If ice alone is used in rather big lumps, the dry bulb thermometer usually remains about 65° F., and the relative humidity deduced from the wet bulb reading varies between 50 per cent. and 70 per cent.[9] The cooling power on

FIG. 2



The portable bed tent in use. The nurse is taking a sample for oxygen analysis.

April 29th, 1932, as determined by Hill's kata-thermometer, was 5·8 with a dry bulb of 66° F., and a humidity of 59 per cent.; while on May 5th it was also 5·8, with a humidity of 69 per cent. Since for comfort the cooling power should not be below 6 in summer or 5 in winter, the cooling power in the tent is reasonable, though it might perhaps be increased with advantage by increasing the radiating power of the tins, though care would have to be taken to prevent the temperature inside the tent from falling too low; further observations are being made on this point.

The tent itself (Fig. 2) is provided with two large celluloid windows in front and two behind. The "sleeve" through which food and drink, &c., are passed is placed towards the front beneath the windows, and below the latter there is, in the more recent model, a piece of rubber tube passing into the tent which can be clipped on the inside; this is to give confidence to the patient, who, if he wished, could take off the clip and breathe through the tube into the outside air; it might also be used for "tube feeding." Dry and wet bulb thermometers (Mason's hygrometer) can be suspended from the roof of the tent, and a piece of lead tubing of very fine bore passes into the tent at the side through which samples of air in the tent are obtained for analysis.

The modified Cecil-Plummer injector which Mr. R. W. G. Davis suggested and has made for me is screwed into the top of a 100-cubic foot oxygen cylinder. From this injector two rubber tubes lead one through the roof towards the bottom of the tent, and the other to the $CO_2$ absorption box; another tube connects the $CO_2$ absorption box with the tent, the whole forming a closed circuit. The injector supplies oxygen to the circuit, and at the same time circulates the air from the tent through the $CO_2$ absorption box and round again to the tent. The rate of supply of oxygen with the corresponding rate at which air is being driven round the circuit is shown by the dial at the top of the injector; it can be varied by turning a handle—this is a new feature. The lower dial shows the pressure of oxygen in the cylinder. The injector is also provided with a by-pass by which the oxygen can be made to pass directly into the circuit without passing through the injector, so that at the beginning of treatment the percentage of oxygen is quickly raised. The by-pass is then turned off, and the injector set to deliver the required amount of oxygen.

It is never necessary to deliver more than 3 litres of oxygen a minute, and 2 or even 1 or 1½ litres a minute are often sufficient to keep up the desired percentage. The economy in oxygen depends on the tent being as air-tight as possible, and to effect this the lower part of the tent should be wrapped closely round the patient's abdomen and kept tight by means of a rubber or elastic bandage at A. One garment at most should intervene between the body and the tent where it is fixed by the belt; the rest of the clothes covering the body are pushed right up into the tent, and the lower part of the body is covered with pyjama trousers and bed-clothes. The extra length of the tent below A is for children who can be pushed right up into it and the opening tied with a piece of bandage. When adults are treated this part of the tent is folded up—hence its shape.

The $CO_2$ absorption box is filled in the upside down position after unscrewing its base. A very efficient mixture to use for absorption is alternate layers of soda lime and flaked caustic soda; the caustic soda is mainly responsible for the absorption, while the soda lime keeps the layers of caustic soda apart and so prevents the flakes from becoming stuck together in lumps. With this mixture it is found that one filling of the tin lasts during several days of continuous use. In my experience soda lime by itself soon becomes exhausted. Messrs. Siebe Gorman supply fused caustic soda on coke, which is also effective; but the tin gets much hotter than with the above mixture. The same firm provides soda lime tinted light green, which turns brown when it becomes exhausted. A window can be provided at the top of the tin near the exit tube, and if a little of this tinted soda lime is poured in first of all some indication may be given when the absorption is giving out; however, so far I have not tried this in practice, but have relied on analysis of the air of the tent for $CO_2$.

Any percentage of $CO_2$ can be used in the tent by altering the by-pass on the $CO_2$ absorption box (C) so that the air flows partly through the box and partly through the by-pass. It is probably the case that $CO_2$ is just as important or more important than $O_2$ in some conditions—e.g., CO poisoning—but our knowledge of its value in pneumonia is still scanty. A very good percentage to try is 40 per cent. of $O_2$ and 2–3 per cent. of $CO_2$. The correct position of the by-pass tap can only be obtained by experimenting. The tap should be arranged in the half-way position in the first place, and after five or ten minutes the $CO_2$ in the tent is determined and further adjustment of the tap made.

Owing to the nature of the cooling and drying arrangements the present oxygen tent is a good deal heavier than those previously described, and it is essential to have a substantial supporting structure. Two patterns have been made. The one shown in Fig. 2 is the portable apparatus; the extension ladders withdraw into a length of 5 ft., and the bar unscrews into two pieces so as to fit on to the luggage carrier of a car. The ice tins are removed, the tent folds up flat round its roof, and can be re-erected anywhere in less than half an hour. But for hospital work a movable trolley which will take the oxygen cylinder and $CO_2$ absorption box and at the same time suspend the tent over a bed would be of undoubted convenience. A trolley is at present in use at Guy's Hospital; but its final form is not yet complete.

When in use the tent requires more or less continuous supervision, at any rate until the nurse is thoroughly familiar with it. The ice tins must be kept filled, and the rate of flow of the oxygen observed from time to time. It is essential to be able to determine the composition of the air in the tent periodically—e.g., at hourly intervals—and this is of special importance if $CO_2$ is being employed. Two pieces of apparatus † have been devised by means of which sufficiently accurate determinations of the $O_2$ and $CO_2$ can be made by a nurse after two or three minutes' instruction. The "clinical oxygen-measuring pipette" is described by Shackle and the author in an accompanying note. The dry bulb thermometer should read about 65° F., and the wet bulb should read at least 8° F. lower. No matches or cigarettes or electrical appliances—e.g., heating pads—are allowed in the tent, and lamps and naked flames must be kept well away from it. Several disasters have been reported elsewhere from neglect of these precautions.

Emphasis has been laid on the tent being air-tight, so as to economise oxygen. No attempt is made to secure this in the American patterns, and Barach [7] has argued that a leak may be of value to ensure the replacement of the rare gases of the atmosphere which may act like vitamins, and may be used up by the patient—both highly speculative propositions as he admits. It is difficult to follow the argument. Some leakage of air into the tent may take place when the sleeve is open in all patterns of tent; but when the tent is closed up the leak will be entirely outwards, and a brisk flow of oxygen will the more quickly drive out the air originally in the tent with its rare gases. Therefore the smaller the leak the more will the rare gases be conserved, because the oxygen formed by electrolysis presumably contains even smaller traces of rare gases than atmospheric air does owing to their slight solubility in water.

### INDICATIONS

So far the tent has been used for some 30 patients at Guy's Hospital and Lewisham Hospital. The results are not yet numerous enough for analysis;

† Demonstrated at the International Physiological Congress, Boston, 1929.

but a few instances of its value in the treatment of pneumonia may be given.

A man, aged 67, with untreated diabetes of several years' duration was admitted to Guy's Hospital on Feb. 16th, 1932, with hypostatic pneumonia and bronchitis. The prognosis was grave. He remained in the tent from 7 P.M. till 10 A.M. the next morning; he was much better, and eventually went out with lungs pretty clear of physical signs.

A baby aged 2, admitted to Guy's Hospital on June 8th, 1932, was very ill with pneumonia. Prognosis almost hopeless. She was put in the tent intermittently for 228 hours; each time she was put in the temperature went down to normal, rising again when she was taken out; she made a complete recovery.

A boy, aged 7, was admitted to Lewisham Hospital on June 2nd, 1932. He was considered "moribund" by the medical officer, and was too ill to protest at being put in the tent at 11 A.M.; at 5 P.M. he had recovered sufficiently to want to come out; he was kept in till 8 A.M. the next day, and on the two following nights for 14 and 15 hours respectively; he made a complete recovery.

I have some evidence that a mixture of $CO_2$ and $O_2$ in the tent may be of value in frequently occurring attacks of asthma; but more observations are required.

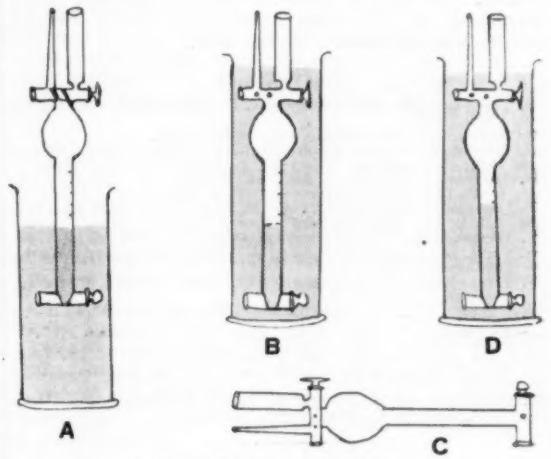
There is already a fairly extensive literature on treatment with oxygen chambers and oxygen tents. Good results, tested during and after muscular work by observations on the respiratory exchange, &c., were obtained by J. M. H. Campbell and myself for chronic bronchitis, emphysema, and myocardial degeneration [5]; we suggested that the same principle was involved in this treatment as in treatment by compressed air which was initiated years ago at the Brompton Hospital. Binger [10] has treated pneumonia patients, and Barach,[11] dealing with 100 patients, claims beneficial results; one patient lived in a tent for two months, which appeared to prolong his life until he had developed immunological resistance to the disease. Barach and Richards [13] obtained good results in congestive cardiac failure, a finding that I can confirm in two cases. Barach [12] has also found that the cardiac pain due to coronary sclerosis was relieved; a patient of mine with frequent attacks of angina found she was better in the tent.

This by no means exhausts the indications for treatment. Since a tent is merely an efficient means of giving oxygen with or without $CO_2$, it should be valuable in carbon monoxide poisoning, shock, post-anæsthetic pneumonia, methæmoglobinæmia, &c.

## DIAGNOSIS

By this is meant the problem of determining when oxygen should be given. It is not a very simple one because, though every one would admit that oxygen should be given when there is arterial desaturation (anoxic cyanosis), there may be conditions when it may be of value even though the arterial saturation is normal. For instance, the heart's action may be so defective that the coronary circulation is slowed, and the muscle is deprived of its normal supply of oxygen, though the local increase of $CO_2$ will enable it to get a larger proportion than normal out of the circulating blood. By giving oxygen this state of affairs may be remedied not so much by the comparatively small extra volume that will be taken up by the arterial blood as by the very much higher partial pressure it will exert. Again, observations by J. M. H. Campbell, G. H. Hunt and myself [14] showed that while decided cyanosis always meant arterial desaturation, the reverse was not always true. In some cases of arterial desaturation there was no cyanosis. Cyanosis is largely a question of peripheral stasis in the parts observed—lips, cheeks, ears, finger-nails, &c. The conclusion is that in all cases of temporary cyanosis oxygen should probably be given; congenital heart disease and polycythæmia are rightly excluded on this definition. But in all cases of breathlessness the administration of oxygen should at least be considered, excluding of course the breathlessness of diabetes and uræmia. When in doubt its effect should be tested, and the best way to do this is to give pure oxygen, using the face mask and large rubber bag that every doctor possesses for giving nitrous oxide gas. If oxygen is given for five or ten minutes in this way the patient will be able to tell if relief is experienced and any improvement in colour may be noticed by the doctor and then a more practicable method of administration may be chosen, and among them the oxygen bed tent should at least be considered.

FIG. 3




A clinical $CO_2$-measuring pipette.

This work was begun under the auspices of the "Clinical Uses of Oxygen" Committee of the Medical Research Council, and the expenses have been defrayed by grants from the Council, to whom my best thanks are due. I should like to thank the sisters and nursing staff of Guy's Hospital and Lewisham Hospital for their willing coöperation and hard work, and the same applies to many of my ward clerks. I wish to thank Mr. A. E. Gould for his help in making the apparatus.

## A CLINICAL $CO_2$-MEASURING PIPETTE

This pipette, shown in Fig. 3, is a modification of Fridericia's alveolar $CO_2$ tensimeter.

It is filled with water by placing it in a cylinder of water; both taps are open as at A. The top tap is then closed and the nozzle of the pipette is connected to the sampling tube of the tent. The pipette is lifted approximately half way out of the water in the cylinder. On turning the top tap the water runs down in the pipette as far as the level of the water in the cylinder drawing air from the tent after it, as shown at A. It is not necessary to take in any exact quantity of air. The top tap is then closed, the apparatus disconnected and lowered again in the cylinder, and left for two or three minutes to equalise the temperature, as shown at B. The volume of air in the pipette is read at the lower level of the meniscus, while the pipette is standing upright in the cylinder. Then the pipette is raised half way out of the cylinder; the funnel is filled with 15 per cent. caustic soda, which is allowed to run slowly into the pipette. The pipette is then further raised from the cylinder and the lower tap closed; the pipette is taken out of the cylinder and turned up and down several times, as at C. The $CO_2$ is absorbed by this means. Care is taken to see that the space below the lower tap is filled with water before the tap is opened beneath the surface of the water, and the pipette is again lowered into the cylinder. After a few minutes to equalise the temperature, the level of water is read again, as at D; the difference between the two readings, without any further calculation, gives the percentage of $CO_2$ in the

atmosphere of the tent. Since the pipette down to the zero mark in the middle of the stem has a volume of 20 c.cm., and the stem is graduated in 10ths of a c.cm., each division will correspond to a 0·5 per cent. $CO_2$. After the determination has been made, rinse through with some water and then with some 1 per cent. sulphuric acid solution to get rid of the alkali.

### CONCLUSIONS

An "oxygen bed tent" is described with the following new features: (1) Cooling and drying are effected by means of ice tins let in through the roof of the tent. (2) Ventilation is produced by means of an injector as described by Cecil and Plummer; but the stream of oxygen with the ventilation can be varied. (3) $CO_2$ is administered by partially short circuiting the $CO_2$ absorption box. (4) A clinical $CO_2$-measuring pipette is described.

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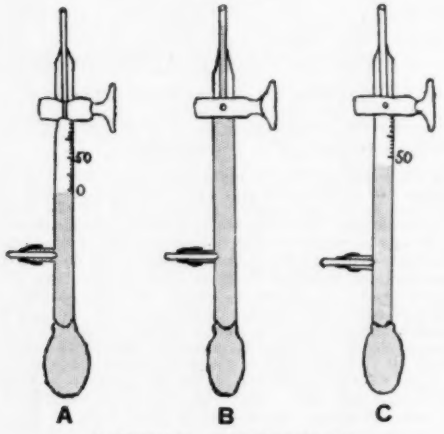
## A CLINICAL OXYGEN-MEASURING PIPETTE

By E. P. POULTON and J. W. SHACKLE

THE top cubic centimetre of the pipette (Fig. 4) is graduated in percentages; the diameter of the tube is chosen so that the zero mark reaches to about 4 cm. below the tap. The rubber bulb has a capacity of ½ oz.

FIG. 4




A clinical $O_2$-measuring pipette

The pipette is filled with a mixture of pyrogallic acid in caustic potash made up as follows: 70 g. of caustic potash in sticks is dissolved in 100 c.cm. water; to this mixture (i.e., 100 c.cm.) is added 20 g. pyrogallic acid. The side tube attached to the lower end of the pipette contains a projecting piece of glass rod fixed to the side tube by a piece of rubber tubing, so that the glass rod can slide backwards and forwards inside the tube. With the tap open, the level of the pyrogallic acid is adjusted to the zero mark by altering the position of the glass rod, as at A. The rubber bulb is squeezed so that the pyrogallic acid runs just above the tap, which is then turned (B).

The nozzle of the pipette is attached to the sampling tube of the tent and the tap opened for one second and then closed. The pipette is then disconnected from the tent and turned up and down until the air bubble ceases to contract. The height to which the level of the pyrogallic acid rises above the zero mark in the pipette gives the percentage of oxygen in the tent. In C this is seen to be 40 per cent. It is better not to allow the bubble to pass into the rubber bulb, as there may be difficulty in getting it back again; to prevent this the pipette can be fixed in a stand which keeps the tap on a higher level than the bulb. This is important because when not in use there should always be about a cubic centimetre of gas in the pipette, and the tap should be closed; if the pipette is always kept in position B the tension-length curve of the rubber bulb becomes altered, so that in a determination less than 1 c.cm. will be sucked into the pipette, and there will be an error in the final reading. The accuracy was within 5 per cent., when tested against a Haldane gas analysis apparatus; this is sufficient for the purpose. The following percentage figures were obtained by J. W. S. (Haldane values in italics): gas mixture A: 57, 55, 56; *54·1.* B: 62, 63, 64; *65·6.* C: 73, 88, 88, 81; *87.*

# Clinical and Laboratory Notes

## AN EASY METHOD OF DEMONSTRATING SOME OF THE VIRUS BODIES

By ALFRED C. COLES, M.D., D.Sc., M.R.C.P. LOND., F.R.S. EDIN.

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IT is now generally, but not universally, thought that virus bodies are living organisms distinguishable from bacteria only by their small size, and, as Prof. J. C. G. Ledingham says, it is a "recently ascertained fact that there exists a group of viruses—variola, vaccinia, fowl-pox, ectromelia, molluscum contagiosum, &c.—whose individual elements are just big enough to be detectable by appropriate staining methods."

The usual method of staining is to fix the air-dried films in absolute alcohol and subject them to long staining with Giemsa, say for 12 to 48 hours, preferably changing the Giemsa two or three times. Incidentally, I may mention that for the last 20 years I have used industrial methylated spirit both for fixing and making up the stains instead of absolute alcohol. It is sometimes advisable to soak the air-dried films in normal saline, Ringer's solution, or even distilled water for ten minutes or so, in order to remove the layer of serum surrounding the bodies and so facilitate the staining; the films are then fixed in alcohol and stained in the usual way. Other methods are first to treat the films with hot Loeffler's mordant and subsequently stain them in gentian or methyl-violet or carbol-fuchsin, as in the process of flagella staining. Personally I prefer the use of Giemsa.

For some time past I have demonstrated these small bodies by simply mixing a drop of the material containing the virus with a drop of Indian ink and spreading a thin film as in Burri's method. Preparations made as suggested can be examined with the

highest powers of the microscope, either by ordinary or by dark-field illumination.

A method I have adopted for some years is to examine, under dark-ground illumination, air-dried, unfixed, unstained, and unmounted smears made on a cover-glass or slide. This very simple and rapid process shows the elementary or virus bodies in many cases exceedingly brilliantly. The method I use is as follows:—

A film of the material containing these bodies is spread not too thinly on a scrupulously clean slide or cover-glass. Allow the smear to dry thoroughly in the air. Examine this smear by any form of dark-ground illumination with an objective whose N.A. does not exceed 0·65. I use a ⅔ inch or 8 mm. apo. objective of N.A. 0·65, and obtain a dark ground by merely turning in an expanding stop below the condenser which is always on the microscope for general use—viz., Watson's parachromatic.

If the air-dried film on the slide be examined without a cover-glass then the tube length of the microscope must be lengthened, this in the case of the Leitz 8 mm. is from the normal tube length of 170 to about 210 mm.; in the Zeiss 8 mm. from 160 to about 185 mm., in order to correct for the absence of a cover. When the correct tube length of a given objective on an uncovered film is once determined it is constant for that objective on any smear notwithstanding the thickness of the slide, whilst the tube length of an object glass varies with the varying thickness of any cover-glass. If, however, a cover-glass is laid on the film on the slide, or if the cover-glass preparation is laid smear downwards on a clean slide, and kept in position by a little plasticine or a spot of melted paraffin, the tube length of the microscope will need very little alteration from that of the normal. Lenses are very sensitive to tube length, especially on dark fields.

Of course the ordinary oil-immersion dark-ground illuminators on the market, when oiled to the slide in the usual way, will give a very bright dark ground, but the simpler method of using a stop of suitable size at the back of an ordinary dry condenser gives a very brilliant image with the ordinary paraffin lamp. The usual ⅔ inch objective found on most medical microscopes can be used if its N.A. is not over 0·65, or it can easily be stopped down to that amount by a funnel or stop behind the back lens, but even then it can only be used when there is a cover-glass on the film, as it will not work through an uncovered film.

The 8 mm. or ⅔ inch objective can be used with any eye-piece from No. 0 to the compensating ocular 18, and I find that either the ordinary No. 4 or the compensating ocular 6 or 8 gives excellent results.

As a matter of fact I can see the virus bodies of molluscum contagiosum well with even a common ⅔ inch of N.A. 0·25 (costing pre-war, 15*s.*) with any of the higher eye-pieces. I have also taken some photomicrographs of these bodies on an old air-dried smear on the slide made three years ago, with an apo. ⅔ inch or 16 mm. of N.A. 0·30 and a compensating ocular 8 giving a camera magnification of 160 diameters. In these photographs the virus bodies come out very sharply.

Films made on slides have many advantages over those made on a cover glass. They are much more easily manipulated, and films of control material may be made by the side of the infected material and so they can be compared; further, if these films are uncovered the position of the best colony of virus bodies, which are often in almost pure cultures, can be marked with a diamond marker and their position logged. Then the slide may be removed, fixed in alcohol, and stained in Giemsa, and the marked organisms can be examined under the 1/12 objective in the usual way.

Permanent preparations can be made from air-dried unstained films by painting round the margins of the cover-glass a ring of melted paraffin and then subsequently a layer of any of the usual cements.

By the above simple method I have been able to examine many of the elementary or virus bodies, among others those of vaccinia, varicella, herpes labialis and zoster, pemphigus, and molluscum contagiosum.* It only takes two to five minutes from the time the smear is made.

When examined in this way the virus bodies in the dry unstained condition appear as distinct sharply defined bright round or oval bodies, singly, in pairs, or in dumb-bell forms, and in short chains, and when experience has been acquired they cannot easily be mistaken, especially when a colony in pure culture is found. So far as I know at present, there is only one possible fallacy; that is, the surface of some glass slides and cover-slips are covered with innumerable roundish minute bodies which on a dark field might be confused with the viruses. This condition cannot be removed by any method of cleaning, and I obviate this by examining the clean slides by dark-ground illumination before spreading the film, and so eliminate any doubtful slides.

Unfortunately I am only able to obtain specimens of the commoner virus diseases, and I should be grateful if some of the workers on these small organisms would kindly spare me any unstained films, or any stained films showing some of the inclusion bodies characteristic of many of these diseases.

* Since the above was written I have confirmed my findings made in 1929 that the virus bodies of foot-and-mouth disease are easily seen in the filtrate by this and other methods. In a paper shortly to be published a description of these elementary bodies with photographs will appear.

## TWISTED OVARIAN CYST WITH ABSENCE OF THE UTERUS

By N. J. Nicholson, M.B. Edin.
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A woman, aged 27, was admitted to the Leicester Royal Infirmary on Sept. 29th, 1932. Violent pain had begun in the right iliac fossa that afternoon, and she had vomited afterwards. The bowels were open satisfactorily, but there had been some pain during micturition on that day. She said she had never menstruated.

She was a pale, thin woman, her tongue was in fair condition, and there was tenderness and rigidity in the right iliac fossa. It was noticed that pubic hairs were absent, but there were sparse hairs on the labia. The vulva appeared normal. The vagina admitted one finger. The top of the vagina felt dome-like, and no cervix could be palpated. The finger felt a rounded swelling in the pelvis not palpable through the abdomen. There was tenderness to the right on rectal examination. Her temperature was 98·2° F., her pulse-rate 96, and her respiration-rate 24.

The patient had a general anæsthetic, and a right paramedian incision was made. A full bladder presented, and a glass catheter was passed. Behind the bladder in the pelvis lay a dark red mass. The upper abdomen was packed off, the patient having been placed in the Trendelenburg position. The mass was brought out of the pelvis; it was roughly spherical, and rather larger than a cricket-ball. It had a pedicle attached to the right side of the pelvis, and the pedicle was twisted two and a half times in a clockwise direction. The twists were undone, the pedicle was clamped and ligatured, and the cyst was removed. Thereafter a better view of the pelvis was obtained. The left ovary was healthy, except for one or two retention cysts in process of extrusion. Associated with it was a structure 1½ in. long, apparently tubal; it had a fimbriated infundibulum, but the ampulla tapered away medially nto the floor of the pelvis and ended about 1½ in. from its ostium. The floor of the pelvis was smooth except for a wrinkle of peritoneum, running from side to side, uniform in thickness. There was nothing suggestive of a rudimentary uterus. On the right side of the pelvis was the pedicle of the cyst which had been removed, corresponding in position to the ovary and tube of the opposite side. The rectum and bladder were healthy. The stump of the pedicle was peritonised, the cysts were removed from the left ovary, the appendix was removed, and the abdomen closed.

This patient has three brothers and four sisters. The sisters menstruate naturally. None of her brothers or sisters have any deformities. The patient herself has had no vicarious menstruation or molimina. Her breasts began to enlarge when she was 16; no enlargement of thyroid or parotid was noticed then. Her breasts were of average size, but the nipples pale and undeveloped. There were no hairs in the axillæ.

The points of interest in the case are (1) the complete absence of the uterus; (2) the influence of this in causing twisting of the ovarian pedicle; (3) the

remnants of incompletely canalised and unfused Mullerian ducts, represented probably by the peritoneal fold across pelvis; and (4) the failure of complete sexual characteristics to develop in spite of the presence of an ovary.

I am indebted to Mr. R. S. Lawson, hon. surgeon to the infirmary, for permission to deal with the case and to publish it.

## IODOFORM POISONING FOLLOWING THE USE OF B.I.P.P.

By R. Cunliffe Shaw, F.R.C.S. Eng.
HON. ASSISTANT SURGEON TO PRESTON ROYAL INFIRMARY

An intelligent girl, aged 8, was admitted to the Preston Royal Infirmary with tuberculous adenitis involving the glands in the submaxillary triangle on the right side of the neck. On April 25th, 1932, at 3.30 P.M., I removed the gland and evacuated the contents of a tuberculous abscess, the floor of which was densely adherent to the internal jugular vein. In dissecting it free the vein was punctured and the lateral ligature applied; apart from this the operation was carried out on the usual lines. The wound was swabbed with ether and smeared with B.I.P.P., the surplus being removed on a dry gauze swab; about 1 c.cm. of the paste was used. The wound was closed with a rubber drain.

The following startling train of symptoms ensued.

At 9 P.M. on the evening of operation there was a pulse-rate of 132, and she appeared somewhat restless, but settled down for a short time after nepenthe ♏ 10. At 6 A.M. the following morning she became suddenly delirious, with a barely countable pulse of 178, and a temperature of 100° F. Camphor in oil was given hypodermically, and the pulse-volume improved. Shortly after this there was an evacuation of bulky stools containing large quantities of mucus and blood combined with a blood-stained vomit. At 9.45 A.M. the pulse was barely perceptible, the respirations 32, of the Cheyne-Stokes type, and the temperature 99°. The child was still delirious, she vomited everything given by the mouth, and there was constant tenesmus and passage of blood and mucus; the skin was hot and dry, but free from any sign of rash.

I must confess that after having used B.I.P.P. so often without untoward result it never occurred to me at this stage that I was dealing with iodoform poisoning. The various suggestions made, such as the infection of the vascular system with a massive dose of tuberculous toxins or the presence of thrombosis and emboli, were easily discounted after further consideration.

The condition reached the crisis at 4 P.M. on the day after operation. The hourly pulse-rate from 10 A.M. to 4 P.M. on this day was as follows: 172, 176, 164, 184, 180, 184, 188. Collapse set in, and the temperature fell to 97°, the fall being accompanied by a low muttering delirium. It was only at this juncture that the diagnosis of iodoform poisoning occurred to me. The wound was opened at both ends, and irrigated with a 10 per cent. solution of sodium bicarbonate, and was noticed that an appreciable amount of iodoform appeared in the washing. Following the irrigations, moist dressings of the same solution were applied. Glucose was given per rectum, and small quantities of arrowroot by the mouth, although the latter were generally rejected. Salines were also given subcutaneously and intravenously.

The results of this treatment were striking; the pulse-rate fell to 132, the respirations became regular and slowed down to 18–20, and the temperature rose to 99°. The stools, although still frequent, became only slightly blood-stained. The irrigations were continued three-hourly for the next two days, and was very noticeable that after the majority of the washouts the pulse-rates showed a considerable fall, whilst during the period before the next irrigation there was a steady rise towards the former level.

On the fourth day there was evidence of a subsidence of the intestinal irritation and a general fall in the average pulse-rate, but a new symptom made its appearance. The child lay in a drowsy state responding only to loud-spoken questions, and giving muttered replies. The pupils were contracted, and she lay in a position of general flexion, the condition resembling the stage of cerebral irritation following the concussion. The stomach was still highly irritable and would retain only small quantities of fluids and semi-solids; vomiting did not cease until the sixth day. During this period the wound irrigation was gradually discontinued and the child was fed with small quantities of sweet fluids, and 20 per cent. of glucose was given per rectum. Her movements in bed were restricted by sand-bags and sheets to prevent her sitting up suddenly, for fear of fatal cardiac syncope. The condition of cerebral irritation gradually subsided after two or three days, and was followed by a drowsy lethargic status, during which she slept most of the day, and from which she slowly recovered to normal mentality.

In this particular case I am convinced that apart from an evident idiosyncrasy to iodoform the symptoms were aggravated by the use of a paste which was too thick in consistency, preventing the spreading of an even film of the emulsion over the wound; in addition, the usage of ether for washing out the wound would also assist in the solution of the small particles of iodoform and their absorption by the tissues.

In looking through the published records, one is impressed by the rarity of reported cases, and by the absence of caution in those people who have advocated the use of iodoform preparations for surgical purposes. For example, Moorhof, in 1905, in advising the use of iodoform bone-plugs, stated that in 220 cases he had never seen one of iodism, and in a girl of 14 he had used 200 g. of his iodoform paste. The cases that have been reported (and they are generally to be found in the literature of the close of the last century and the first decade of this) fall into three groups:—

(1) Those which exhibit cutaneous eruptions only.
(2) Those where psychological phenomena were the principal symptoms.
(3) Those which exhibit a dramatic constitutional reaction characterised by: rapid pulse-rate, high temperature and dry skin, vomiting and diarrhœa, great restlessness and delirium, and rapid loss of weight.

It is suggested that this latter acute type of iodoform poisoning is due to stimulation of the thyroid gland, resulting in the production of a temporary thyroid toxæmia. The case I have recorded here undoubtedly belongs to this group. It is noteworthy because it shows, firstly, the vital necessity of a correct diagnosis, and, secondly, the speedy response to treatment by irrigations, salines, and glucose.

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North of England Obstetrical and Gynæcological Society.—At a recent meeting of this society, held in Manchester, Dr. F. H. Lacey delivered a presidential address on Cæsarean Section. He began by tracing the history of the operation from Roman times, and by describing early operations, one by Barlow of Bolton in 1793, and one by Trantma of Willenberg in 1600. The reported mortality of 84 per cent. from 1737–1860 was reduced, he said, by Porro in 1877 as a result of supravaginal hysterectomy being also performed. After referring to Sanger's operation in 1882, and Osiurder's lower segment operation in 1865, he quoted more recent figures such as those collected by Routh (6·1 per cent. in 602 cases for 1906–1910) and Spencer (maternal mortality 3·3 per cent., and infantile mortality 10 per cent. in 120 cases). The reasons, he thought, for the present abuse of the operation were: (1) demand on the part of the patient for a shorter and less painful labour; (2) an exaggerated idea of the value of the fœtal life as compared with that of the mother; and (3) extension of indications to include the convenience of husband, patient, or doctor. The lower segment operation was preferable if the patient was in labour or was potentially septic, and when this variety of operation was performed the transverse incision was desirable. Dr. Lacey then gave an analysis of 200 of his recent hospital operations. There were only two deaths in the series—one from broncho-pneumonia and the other in a case complicated by many adhesions which followed two previous Cæsarean sections. One operation, he said, did not preclude a subsequent normal confinement. Cæsarean section for eclampsia, although not popular in this country, was apparently more successful on the continent. Open ether was the anæsthetic used in the majority of cases, but spinal and other special forms of anæsthesia might be strongly indicated in such conditions as toxæmia of pregnancy.

# MEDICAL SOCIETIES

## TUBERCULOSIS ASSOCIATION

AT a meeting of this association, which was held in London on Jan. 20th, Prof. S. LYLE CUMMINS spoke on

### Tuberculosis and the South African Native

He said that to the student of tuberculosis the South African Tuberculosis Report [1] presented most interesting problems. One of these was the relation between the extreme tuberculosis susceptibility observed by Borrel in the Senegalese levies brought to France for the war and thus brought into contact with tuberculous infection for the first time, and the almost equally marked susceptibility of the native miners on the Rand, recruited from the territories where tuberculosis was now known to be endemic. The Borrel type might be correctly described as the tuberculosis of virgin soil, while the South African type would be better called the tuberculosis of hyper-allergic soil. The very extensive and important tuberculin tests carried out in the course of the South African investigation proved that those African mine recruits were not merely tuberculin-sensitive, but were so markedly sensitive as to present a new phenomenon in tuberculo-allergy. Whereas in Wales only 2·3 per cent. of adult males reacted to an intradermal test of 1 in 10 million and only 5·5 to 1 in 1 million dilution of tuberculin, the corresponding figures for the South African mine recruits were 48·6 and 51·5, according to the observations recorded in the report. These figures justified the application to the tuberculosis of these persons of the term "tuberculosis of hyper-allergic soil." Follow-up work had proved quite definitely that these hyper-allergic persons were liable to acute fatal tuberculosis very similar to that observed by Borrel in non-reactors; and, further, that their liability varied directly with the severity of their reactions to tuberculin. Those natives whose reactions were noted as "positive plus" on arrival were found to be more liable to develop tuberculosis during subsequent mining service than the natives who were merely recorded as "positive," and the latter were more liable than those classified as "negative." Prof. Cummins pointed out, however, that the dilution of tuberculin used for these tests was very high—1 in 5000; observations in Wales had shown that no less than 62·5 per cent. of adult males who were negative to 1/5000 tuberculin were positive to 1/1000, 87·5 per cent. to 1/100, and 99 per cent. to 1/10. Thus the "negative" category in the South African tests must have included a large number of those who would have been reckoned positive if more concentrated tuberculin had been used. This South African group might also have included a certain small proportion of genuine "virgin-soil" natives of the Borrel type, so that the number of positives included in the negative group could not be deduced from any analogy with the figures from Wales. There was evidence that the Borrel type of "virgin-soil" susceptibility was not yet entirely lost as a phenomenon of the industrialisation of African natives. The prevalence of the "hyper-allergic" type was, however, of greater practical importance, since this implied that contact with subinfections had failed to produce in these natives any degree of tuberculo-resistance comparable to that of Europeans.

[1] See THE LANCET, Jan. 14th, p. 100.

#### ACQUIRED RESISTANCE

Were they to infer from this that the theory of acquired resistance to tuberculosis resulting from subinfection was untenable, and that some other explanation of the relative immunity of Europeans as compared with Africans must be sought for? Prof. Cummins held that such an inference would be rash. In experimental tuberculosis, the fact that a previous mild infection conferred marked relative immunity to subsequent reinfection was established. Even among "hyper-allergic" Africans, clinical observations proved that the long-service natives, exposed to occasional infection over long periods, and at last developing the disease, tended to possess a greater power of controlling their temperature and of keeping their systemic disturbance within bounds than those natives who developed their tuberculosis earlier in their mining service. What, then, of the increased liability to develop tuberculosis which had been shown to go with markedly positive tuberculin reactions in these "hyper-allergic" mine recruits? How did it happen that the increased exposure to infection which must presumably underlie these "positive plus" reactions led, in the case of recruits, to increased liability, while the prolonged opportunities for getting infected which appertained to long mine service led, not to increased liability but to increased toleration and increased resistance? The answer would appear to be that exposure to heavy infection under tribal conditions was one thing, and prolonged exposure to casual infection on the mines quite another. The first led to the establishment of larval but often active lesions which, while producing marked allergy, were very liable to flare up into generalised disease; the second led on to the development of some degree of resistance so that the disease when it developed tended to assume a more benign character. The lesions which underlay the hyper-allergic type in these natives were apparently impossible to detect by routine physical examination, and were probably situated in intrathoracic and intra-abdominal glands, where they escaped diagnosis. It might be assumed that the more severe reactions were associated with a greater liability to disease because they were the result of heavier infections and more active lesions. The less reactive groups owed their diminished risk to the fact that in them the lesions of previous infection were of smaller extent and less activity, calculated to lead to a slighter degree of allergic potential. The "positive plus" reactors possessed what Prof. Cummins had described as an "acquired liability" to the disease.

#### ALLERGY DISTINCT FROM IMMUNITY

If this concept were accepted, then the important biological deduction followed that, in tuberculosis, allergy could be distinct from immunity. It was clear that these natives, exposed to infection under tribal conditions, responded not so much by the production of tuberculo-immunity as by the development of extreme tuberculo-allergy, and though it was true that this hyper-allergic phase might be an early stage on the road to resistance, it was not the same thing as resistance, nor was it even accompanied by any appreciable amount of resistance to the disease. Its development would appear to render the individual liable to the toxic phenomena of over-severe reactions to auto-tuberculin which resulted in acute and fatal symptoms when localised lesions became generalised under physiological stresses.

Under tribal conditions the African appeared to tolerate tuberculous infection. It was only when he was exposed to new and trying conditions that his larval lesions showed a tendency to sudden generalisation, and to pass into a type of disease so rapidly fatal that the average period of survival in the mine hospitals was only about 33 days.

The African native seemed to differ from the European in his response to tuberculous infection, and his inadequacy might, perhaps, be similar to that manifested by the special strains of guinea-pigs produced through in-breeding by Lewis and Loomis. Tuberculosis had probably become endemic in the native territories of South Africa only recently, and a much longer time and many generations might be necessary for the acquisition of enhanced powers of developing resistance by African natives.

Prof. Cummins concluded by urging that the African native, with his marked liability to tuberculosis as well as to pneumonia and other inspiratory infections, must be housed, fed, and cared for on expensive and generous lines.

At the evening session Dr. P. ELLMAN, tuberculosis officer, East Ham Tuberculosis Dispensary, read a paper on

## Pulmonary Asbestosis

He spoke of the clinical, radiological, and pathological features of the condition, and of the risk of phthisis involved. He said that recent advances in their knowledge of the pneumoconioses had enabled research workers to find out certain characteristic features common to certain industries, the greater part of the work having centred round the subject of: (*a*) silicosis, depending on the presence of free silica; and (*b*) the silicatoses, arising from the inhalation of various dusts of fibrous minerals of the silicate group, including asbestos. Pulmonary asbestosis had come to be recognised as a distinct clinical entity, in which, owing to the diffuse nature of the resulting fibrosis, the degree of respiratory embarrassment was usually greater than that of an equally advanced case of silicosis, and which was in fact a more severe disease than the latter.

After a short historical survey of the development of a knowledge of pulmonary asbestosis as a clinical entity, and a review of the chemistry and industrial uses of asbestos, the speaker went on to describe the clinical features of pure pulmonary asbestosis.

### CLINICAL FEATURES

The onset of symptoms and their severity depended to a great extent on the nature of the work and of the amount of dust inhaled. Symptoms might be altogether absent, despite the presence of clinical and radiological signs. In Dr. Ellman's experience, dyspnœa was one of the earliest symptoms which at first only occurred on exertion but which became progressive until ultimately dyspnœa and even orthopnœa might occur, and not only after effort. A harsh, non-productive cough and fleeting chest pains would sooner or later accompany the dyspnœa. Expectoration was often absent, and when present was usually scanty in amount and of a tenacious glairy mucoid nature. In the winter months the sputum was usually more abundant and often more purulent. True hæmoptysis had been absent in all his cases, even blood-stained expectoration having been exceptional. Night sweats and giddiness were occasionally found. Anorexia might occur early, but was usually a feature of the more advanced cases, this also being true of cyanosis. Emaciation out of all proportion to the physical signs had mostly been a distinctive feature. Finger clubbing was present in the more advanced cases where bronchiectasis was usually present. A characteristic skin lesion on the hands or legs, known as "asbestos corns," was complained of by many of his patients. While most of the symptoms enumerated were common to silicosis, the more distinctive features of asbestosis were the cyanosis and dusky complexion, anorexia, and emaciation and loss of weight out of all proportion to the physical signs. Examination of the chest showed a very limited chest expansion, especially at the bases where the fibrosis began. The apices were usually retracted. The percussion note was impaired, particularly at the bases. Auscultation showed diminution of breath sounds. The adventitious sounds—fine, dry, crackling râles—were characteristic of the disease. The physical signs, in short, were those of a basal fibrosis, usually spreading upwards. Radiological examination showed in the later stages a fine diffuse punctate mottling (in marked contrast to the coarse nodular mottling seen in silicosis), confirming the clinical evidence that the disease was characterised by a fine diffuse fibrosis beginning at and involving principally the lung bases.

### PATHOLOGICAL FEATURES

The macroscopic appearance of the lungs and pleura showed that the visceral pleura was usually markedly thickened, and that the pleural sacs might be partially or even completely obliterated by dense fibrous adhesions. Microscopically the essential features of pulmonary asbestosis were: (1) a chronic interstitial fibrosis of the lung, and (2) the presence of characteristic asbestosis bodies within the alveoli and embedded in the fibrous tissue either separately or in clumps. The interstitial tissue was everywhere increased, so much so that in some cases the alveolar structure could no longer be detected, while in other areas there was evidence of compensatory emphysema. It was in and around the densely fibrosed areas that the bodies were found in the greatest profusion, and tended to occur in radially arranged clumps. Asbestosis giant cells, first described by S. R. Gloyne, might be present in the connective tissue. This giant cell was larger than the tuberculosis giant cell, and the cytoplasm had a stippled and pigmented appearance compared with the structureless caseating appearance of the tuberculosis giant cell. Asbestosis bodies—the highly characteristic golden yellow bodies found in the sputum and fibrosed lungs of asbestos workers—had been found to be present in autopsies of all cases of pulmonary asbestosis. In the sputum these bodies usually occurred singly or in groups of two or three, but when they were more numerous they might be seen in radially arranged clumps—an occurrence indicative of disintegration of lung tissue, either by simple suppurative broncho-pneumonia or by secondary tuberculous infection. The mere presence of these bodies in sputum, while they would indicate exposure to asbestos dust, could not alone justify a diagnosis of pulmonary asbestosis. The diagnosis depended on the cumulative findings: (*a*) opportunities for inhaling asbestos dust; (*b*) clinical evidence of a pulmonary fibrosis; (*c*) radiological evidence of a fine, diffuse pulmonary fibrosis; (*d*) asbestos bodies in sputum, fæces, or lung tissue. It was remarkable for how long asbestosis bodies persisted in the sputum, even when the period of exposure had been very short. In one case exposed to the dust for only ten weeks the bodies were present in the sputum nearly five years later, suggesting that once the bodies were formed they remained more or less permanently in the lung.

With regard to prognosis Dr. Ellman said that an interesting feature of the disease was the extreme length of time that elapsed as a rule between the exposure to the dust and death. The disease was progressive even after cessation of work, and there could be no doubt that without adequate protection the prognosis in pulmonary asbestosis was grave, the gravity varying directly with the degree of concentration of the dust to which the worker was exposed. The first symptoms of the disease were observed, as a general rule, from 4 to 20 years after exposure, the time depending upon the nature and concentration of the dust. He briefly outlined preventive measures before passing on to the question of

TUBERCULOSIS RISK IN ASBESTOSIS

Until quite recently it had been assumed that it was comparatively rare for a pure asbestosis to be complicated by phthisis, and some authorities still maintained that the effect of asbestos and the lung changes induced thereby were due to silica. The constant findings of the asbestosis bodies in the lungs, however, apart from anything else, refuted such an idea. The effect of inhaled dust on the development of a pulmonary infection, especially tuberculosis, was of great clinical importance. Dr. Ellman referred to E. H. Kettle's observations on various dusts, and his classification of them into active or inert substances. Both silica and asbestos came into the group of active dusts, producing active lesions in the subcutaneous tissues as well as a serious pulmonary fibrosis. Kettle had also found that the active dusts had a definite influence on a tuberculous infection, assisting the growth of tubercle bacilli. In Dr. Ellman's own series of 17 cases, the majority of whom were not at work, six had tuberculosis in addition to asbestosis, the disease being active in four of these. That there was a definite risk of tuberculosis in asbestosis appeared to be beyond a doubt, but further work would have to be done before the degree of risk could be assessed. Existing uncomplicated cases would have to be observed, and their development followed by repeated clinical and sputum examinations and serial skiagrams.

## LIVERPOOL MEDICAL INSTITUTION

AT a pathological meeting of this institution held on Jan. 19th, with Mr. F. STRONG HEANEY, Vice-President, in the chair, Prof. M. J. STEWART (Leeds) read a paper on

### Silicosis and Allied Diseases of the Lungs

He said that the term anthracosis should be reserved for conditions resulting from the inhalation of carbonaceous dust, which produced a fibrosis of the lungs limited both in severity and extent. It did not conduce to tuberculosis. Dense or extensive fibrosis in coal miners was due to an accompanying or antecedent silicosis, and was met with chiefly among anthracite miners. He pointed out the difference in the gross and microscopic anatomy of silicosis and asbestosis; the former was nodular, whorled, mainly central in distribution, and involving especially the middle and upper parts of the lungs; the latter diffuse and mainly basal. The tendency to tuberculosis was significantly less in asbestosis but none the less very important. Asbestosis was definitely a more serious disease, proving fatal on an average after 13 to 15 years' employment as compared with 32 to 42 years in silicosis. Prof. Stewart gave some account of the morphology, distribution, and mode of formation of the asbestosis body, and discussed the significance of its presenec singly or in clumps in the sputum. He described the gross silico-siderotic lesion of the lungs met with in hæmatite miners in the West Cumberland ironstone field, and suggested that the victims of this severe disease were mainly those men who were employed on the dry mechanical drill from the time of its introduction in 1913 until its replacement by the wet drill a few years ago.

DISCUSSION

In the discussion which followed, Prof. J. H. DIBLE asked Prof. Stewart what was his experience with the polarising microscope in silicosis cases, as it appeared that in many cases of undoubted silicosis, with typical nodules, no doubly refractile bodies could be demonstrated. It seemed difficult to believe that these became dissolved subsequent to the formation of the nodule. Might the resistance of animals to pneumoconioses be due to the greater complexity and length of their upper air-passages? Man was not only less well protected in this respect but was also to some extent a mouth-breather; and these two factors contributed to his liability to respiratory disease. The toxicity of dissolved silica was definite; acute lesions such as fatty degeneration of the heart and necrosis of the liver might be produced by it. Prof. Dible agreed with Prof. Stewart that there was rarely any real difficulty in distinguishing silicotic lesions from fibrous lesions of tuberculosis.

Prof. J. M. BEATTIE considered that experimental work on silicosis should, if possible, be carried out by the introduction of the dust into the lungs by inhalation. The fibrous overgrowth could be produced over a comparatively short time if the dust concentration was sufficient, and if each animal was compelled to breath the dust continuously for one or two hours daily. He showed photographs in support of his contention that conditions similar to those seen in early cases of silicosis could be reproduced in guinea-pigs by the inhalation method. He still believed that the irritative action of the particles played some part in the process. In his view the prevalence of tuberculosis in cases of silicosis was due to neglect of sanitary precautions rather than to a predisposition to infection with *B. tuberculosis*.

Dr. R. H. MOLE asked why asbestosis affected mainly the lower lobe of the lung, and silicosis the upper and middle areas.

Dr. C. ALLAN BIRCH asked why tuberculosis was less frequently associated with asbestosis than with silicosis.

Dr. C. O. STALLYBRASS referred to the frequency with which silicosis turns up in occupations in which the risk would not have been anticipated, such as in cotton carders and men engaged in polishing the heels of boots. He discussed the greatly increased danger of an associated inhalation of silica with a simultaneous infection of tuberculosis.

Dr. R. W. GEMMELL said that radiologically the differential diagnosis between silicosis and tuberculosis was often impossible. It was very difficult to say where silicosis ended and tuberculosis began. It was difficult to get a history from masons, who usually denied having hæmoptysis and cough for fear of losing their job.

REPLY

In replying to the points raised in the discussion, Prof. STEWART said that undoubtedly solution of silica particles went on in the lung substance; it was the soluble silica which produced the pathological defects. Optically active particles slowly disappeared from silicotic nodules. Locally, colloidal silica

produced an intense coagulated necrosis followed later by endothelial cell reaction and proliferation of fibroblasts round the lesion. In rabbits and guinea-pigs asbestos fibres certainly got into the lungs and produced asbestosis bodies, but the pathological changes were slight. He was unaware of any statistical evidence showing that cancer of the lung or bronchus was particularly common in cases of pulmonary fibrosis due to dust. He had seen at least two cases with that association. It was interesting that in animal experiments it was not possible to produce pulmonary fibrosis with ground-up asbestos. He had seen acute silicosis in (1) sandblasting; (2) the making of abrasive soaps and powders. The lesion here still tended to be focal but did not present the densely collagenous whorled character of chronic silicosis; it was rather an active fibroblastic proliferation perivascular in distribution. He was interested to hear that in Sheffield Prof. Beattie had seen only two cases of silicosis in knife-grinders in whom tuberculosis was *not* present. He agreed that Dr. Bridge's figure, 56·6 per cent., was probably an under-estimate. He thought that the tendency for pulmonary fibrosis in asbestos workers to be mainly basal was due to the character of the dust. It consisted of fine fibres of varying length, and it was probable that the action of gravity played an important part in getting them well into the parenchyma of the lung. They could only reach the lung alveoli by the fibre passing end-on. It was very difficult to have systematic X ray examination of any particular group of workmen, as they were liable either to be scared and think they would lose their job, or get inflated notions as to the possibility of compensation. Tylecote and Dunn, and W. E. Cooke, of Wigan, described bodies somewhat resembling asbestosis bodies in the lungs of coal miners who had never been near an asbestos factory. One reason why tuberculosis was more prevalent in cases of silicosis than in cases of asbestosis was that the latter disease was much more rapidly fatal; another explanation was the insanitary habits of knife-grinders. Silicosis was a disease of males essentially, asbestosis largely a disease of females. The general hygienic conditions in asbestos factories were probably pretty good. He was interested to hear Dr. Stally-brass's opinion that silica rather than silicon was the danger. It was probably a question of solubility. Carborundum (silicon carbide) was found by E. H. Kettle to be almost inert on local inoculation. He was interested to hear of the frequency of pulmonary fibrosis among the masons engaged on the Liverpool Cathedral and the drillers and quarriers in the Mersey Tunnel.

## LONDON CANCER SOCIETY

THIS society, which was recently formed, held its inaugural meeting, under the presidency of Mr. J. P. LOCKHART-MUMMERY, on Jan. 27th at 144, Harley-street, W.

Mr. T. YOUNG SIMPSON, hon. sec., and Dr. P. CAVE, hon. treas., explained that the objects of the society were to study, as far as possible, every aspect of the cancer question and to report on new methods of treatment and new pathological and surgical methods of diagnosis with a view to the discovery of some common ground of progress. They appealed strongly to the profession to attend the meetings and to bring forward any new idea, improvement in technique, or practical discovery which might suggest a new avenue of exploration or assist in alleviating or curing any form of malignant disease. There was already a feeling amongst the more sanguine members that before long cancer might be treated without some of the more serious surgical methods now in vogue and possibly by quite simple medical means.

Dr. PAUL CAVE read a paper, illustrated by radiograms, on Osteoplastic Metastases in Cases of Cancer of the Prostate.

In one of his cases a man, aged 50, with no initial symptoms of prostatic trouble, suddenly developed backache. X rays showed extensive osteoplastic deposits in the lower dorsal, lumbar, and sacral vertebræ. It was then discovered, on routine examination, that the prostate was hard and knobby. Complications developed rapidly, including incontinence of urine and fæces, paralysis and anæsthesia of both legs—all symptoms of pressure upon the spinal cord. The cervical and upper dorsal vertebræ and the skull were not affected.

Autopsy revealed a stony-hard condition of the affected vertebræ, the chemical composition of the bone showing a 44 per cent. increase in the calcium carbonate content. The marrow spaces were shown on decalcification to be filled with solid masses of epithelium. Practically no marrow remained and the extension of the growth into the spinal canal had severely compressed the cord. The growth (an adenocarcinoma) in the prostate extended into the bladder and pelvic tissues. There was no rheumatic history and the Wassermann reaction was negative. The lungs, liver, kidneys, and spleen were normal. There was no affection of skull, brain, or meninges.

Dr. Cave said that it was difficult to explain why sclerosis should develop in bone instead of osteoporosis, and why only the lower spinal vertebræ should be affected. If the sclerosis of bone was an effort of nature to strangle the growth why did it occur specially in cases of prostatic cancer and not in all cases of bone metastases. A possible explanation was: (1) spread from the primary growth through the lymphatics, whereas osteoblastic metastases follow vascular dissemination; (2) cancer cells invade the bone through the Haversian canals and lymphatic channels after the ligaments and periosteum have been infiltrated; (3) the various blood-vessels become choked with cancer cells and so the blood-supply of the parts is diminished; (4) this restricted blood-supply deprives the bone cells of parathyroid secretion, causing retention of calcium in the form of calcium carbonate; (5) the new bone encroaches on the Haversian canals and medullary spaces and makes the bone abnormally dense and hard.

Dr. MAURICE SHAW read a paper on Some Medical Aspects of Cancer. He held that it was an advantage for those who suffered from apparently innocent complaints to submit themselves to a thorough and complete medical examination from time to time in order to free themselves from the subconscious bogy of cancer. He said that often patients improved in health when a fear to which they would not give expression in words was dispersed by a definite statement excluding any form of malignant disease. He expressed his confidence that by team-work it was now possible to verify or exclude the majority of cancerous tumours, but conceded that up to the present no serum test for the presence of cancer in the system had proved efficient. In his view intrathoracic cancer, growths in the large bowel, malignant prostate, and so on, were sometimes missed, because trouble was not taken to investigate matters more thoroughly; he urged practitioners to keep abreast of the times. He believed that a society such as the London Cancer Society could do much good by hearing the views of experienced clinicians, surgeons, pathologists, and laboratory workers, from a careful sifting of which definite advances might be made.

Dr. DAVID DE SOUZA, Mr. MCNEILL LOVE, Dr. C. E. M. LOWE, Dr. S. W. H. STUART, and others took part in the ensuing discussion.

# REVIEWS AND NOTICES OF BOOKS

## Gray's Anatomy

Twenty-fifth edition. Edited by T. B. JOHNSTON, M.B., Ch.B., Professor of Anatomy in the Medical School of Guy's Hospital. London: Longmans, Green and Co. 1932. Pp. 1478. 42*s*.

IT is no surprise to learn that the last edition of this famous work was so favourably received that the present issue was called for earlier than had been anticipated. We have little doubt that the history will repeat itself. It is a real pleasure to read the descriptive accounts of bodily structure in this book. The editor has rewritten a good deal of the section on surface anatomy, and has incorporated in the embryology section many of Prof. J. E. Frazer's recently published views; otherwise the main changes have been in small revisions and in the addition and replacement of figures. It is to be hoped that Prof. Johnston's ardent wish may be fulfilled, and that the questions of terminology which have been upsetting the anatomical world for so many years will soon be settled to the satisfaction of English anatomists. The amelioration of the student's lot, in this respect, produced by the simple use of English equivalents whenever possible, is a move in the right direction which is here adopted. A fine book, commendable in every way.

## The Colon, Rectum, and Anus

By FRED W. RANKIN, M.D., F.A.C.S., Associate Professor of Surgery, the Mayo Foundation; J. ARNOLD BARGEN, B.S., M.D., M.S., F.A.C.P., Assistant Professor of Medicine; and LOUIS A. BUIE, M.D., F.A.C.S., Associate Professor of Proctology at the Foundation. London: W. B. Saunders Co., Ltd. 1932. Pp. 846. 50*s*.

THIS authoritative work contains a historical account of the diagnosis and surgical treatment of disease of the large bowel and rectum, and incorporates a record of the clinical experience and statistical data accumulated at the Mayo Clinic. Certain sections of the book demand especial notice. The chapter on megacolon gives a complete summary of the modern view of the pathology of this condition, and an indication of the hopeful results which are being obtained by sympathectomy. The long chapter on chronic ulcerative colitis provides a masterly exposition of the subject. Bargen's diplococcus is stated to have been isolated in pure culture from approximately 80 per cent. of patients with chronic ulcerative colitis examined at the Mayo Clinic during recent years, and a similar organism has been obtained by blood culture from certain fulminating cases of the disease. It is not believed at this clinic that the dysentery bacillus plays any part in the immediate ætiology. The incidence of complications, such as stricture, malignant disease, and peritonitis, is discussed, and the treatment is described in a helpful way. A large portion of the book is naturally devoted to the subject of carcinoma of the colon and rectum. Surgical excision is considered to be the method of choice in dealing with malignant disease of the rectum; the patient's condition, together with the site and characteristics of the growth, being considered in determining the most suitable surgical procedure. A cautious attitude is adopted as to the value of radium in rectal cancer. The final chapter on operative procedures contains many features of interest, including the methods of resection of the colon with aseptic anastomosis, and Rankin's method of "obstructive resection." A two-stage abdomino-perineal excision is described, the excision operation being started from the perineum at the second stage. For the treatment of megacolon the method of exposure and division of the presacral and inferior mesenteric nerves is described.

The bibliographies given at the end of each chapter show what an immense amount of original research has been done in this branch of surgery at the Mayo Clinic. The volume forms an impressive and valuable monument to the work of the authors and their colleagues, as well as a critical and up-to-date book of reference on the colon and rectum. It should be widely studied.

## The Principles and Practice of Psychiatry

By ALEXANDER CANNON, M.D., and EDWARD DUNCAN HAYES, M.D. London: William Heinemann, Ltd. 1932. Pp. 437. 25*s*.

THIS work is an attempt to write a compendium of modern psychiatry for the use of students. While the divisions and proportion of the work are on the whole adequate, the same cannot be said of the subject matter which is presented in an undigested and fragmentary form. At places, owing to the lecture-note style, the statements are incoherent, or, by omission of relevant data, misleading. After an introductory chapter on general principles the forensic and criminal aspects of psychiatry are considered, followed by sections on mental deficiency and abnormalities of behaviour in children. Psychology is the subject of the next three chapters; then systematic psychiatry is dealt with, especial attention being given to the endocrine glands in this connexion. The application of physiology to psychology and of biochemistry to psychiatry are discussed at some length, and a chapter on methods of examination concludes the book, except for an appendix which is a medley of tables of doses, psycho-analytical symbols, anatomical diagrams, classification lists, and legal forms and other odds and ends, including the deaf and dumb alphabet. The production, for a text-book, is too uncritical and subjective; this is particularly evident in the section on psychotherapy. The book seems to be compounded of facts collected without due sifting from many sources, and of personal views which require more support before inclusion in a text-book for students.

## The Failing Heart of Middle Life

By ALBERT S. HYMAN, A.B., M.D., F.A.C.P., Cardiologist, Beth David and Manhattan General Hospitals; and AARON E. PARSONNET, M.D., C.M., F.A.C.P., Attending Physician and Cardiologist, Newark Beth Israel Hospital. Philadelphia: F. A. Davis Company. 1932. Pp. 538. $5.00.

UNDER an attractive title Dr. Hyman and Dr. Parsonnet deal in great detail with the various types of heart disease which result from lesions of the coronary arteries. The first six chapters are devoted to an anatomical, physiological, and pathological introduction to the subject, and the next five to the syndrome of "myocardosis," this term being applied, instead of the more familiar title of myocardial degeneration, in lesions other than the inflammatory ones resulting from myocarditis. Three sections of

the book are devoted to coronary occlusion, to the electrocardiographic changes in coronary disease, and to angina pectoris; while in a final section the authors deal with medico-legal problems arising in cases of sudden death.

The importance of the great degenerative group of cardiac lesions needs no emphasis, for the advances in our knowledge of the clinical manifestations of coronary occlusion in the last 20 years only serve to emphasise the extent of our ignorance concerning other types of angina. As a book of reference this will be welcomed by all who are trying to explore this field; but at the same time one cannot but regret that the authors have in places lost sight of their objective. While the chapters dealing with pathology are to the point and are well illustrated by microphotographs, those on the clinical aspect are at times unnecessarily verbose and treat in great detail topics which should already be familiar to all readers whose clinical training is not entirely negligible. Similarly in the section on electrocardiography six chapters are devoted to disturbances of the heart's rhythm. While it is recognised that almost every type of abnormal electrocardiogram may be met with in the degenerative group of cardiac diseases, this is surely not the place to discuss the symptomatology and diagnostic features of the various types of arrhythmia. The reader who is prepared to plod through a book of over 500 pages on coronary arterial disease deserves to be given credit for at least an elementary knowledge of electrocardiography. In a future edition much padding might with advantage be omitted. A minor point is that the lettering of the electrocardiograms is not that commonly adopted, the initial major ventricular deflexion being marked R irrespective of whether it happens to be positive or negative. The medico-legal section is a useful innovation, but would be easier to digest if more space were devoted to a discussion of the legal principles involved even if fewer actual cases were quoted to illustrate these principles. The bibliography includes 1250 references but even so appears to be incomplete, several original papers which are referred to in the text being omitted.

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### Journal of Pathology and Bacteriology

THE January issue (Vol. XXXVI., No. 1) contains the following papers. Familial and Bilateral Tumours of the Carotid Body, by W. H. Chase (Montreal). Two sisters with tumours of the carotid body, in one of them on both sides. They were removed and one of the women died. The tumours had the typical structure of the carotid body; there were no metastases and no general symptoms were identified.—On the Significance of Nerve-fibres in Human Malignant Neoplasms, by G. M. Ryrie (Glasgow). The nerve-fibres found in carcinomata are not proper to the tumour, but axis cylinders sprouting from the normal nerves injured by the tumour as they do from after any nerve section.—Experimental Liver Disease Produced by X Ray Irradiation of the Exposed Organ, by A. Bolliger and K. Inglis (comment on p. 258).—The Pathological Action of Light: "Light Stroke" and its Relation to "Heat Stroke," by A. G. Levy (London). Mice, sensitised with hæmatoporphyrin and exposed to visible light, die with symptoms very similar to those of heat stroke. Intense irradiation with ultra-violet light does not kill depilated mice if they are kept cool.—Neoplasms in Fish: a Report of Six Cases with a Summary of the Literature, by A. Haddow and I. Blake (Edinburgh). Lymphosarcoma, hepatoma, ganglioneuroma, melanoma, &c.—The Filtration of Herpes Virus through Graded Collodion Membranes, by W. J. Elford, J. R. Perdrau, and W. Smith (London). The diameter of the virus particles is about 0·1 to 0·15 μ.—The Production of Immunity to Vaccinia Virus by Mixtures of Immune Serum and Virus, and the Importance of Phagocytosis in Antivaccinial Immunity, by R. W. Fairbrother (Manchester). Intracerebral inoculation of virus serum mixtures leads to a high degree of immunity; immune blood has more action on the virus than immune serum.—Observations on Salmonella Agglutination and Related Phenomena, by P. Bruce White (London). For experts by an expert.—Species Immunity to Pneumococcus, by H. B. Day (London). In mature pneumococci an antigen is formed which on injection into animals induces immunity to all types of pneumococci; the serum confers a similar passive immunity. The type-specific antigens are formed by young pneumococci, and the more generalised species antigen appears to be derived from them by dissociation of the type-specific substance.—The Production of Staphylococcal Hæmolysin with Observations on its Mode of Action, by J. W. Bigger (Dublin). The production of hæmolysin is inconstant and the best conditions vary with different strains; glycerin and phosphate are useful.—Reticulo-endotheliomatosis, Ovarian Endothelioma and Monocytic (Histiocytic) Leukæmia, by R. Gittins and J. C. Hawksley (Birmingham). In an infant of one year, with a good discussion of the literature.—Sarcosporidiasis in Human Cardiac Muscle, by J. A. Hewitt (London). Accidentally found in histological material.—Production of Tumours in the Fowl by Carcinogenic Agents: (1) Tar, (2) Dibenzanthracene, by P. R. Peacock (Glasgow). Both agents produce tumours on subcutaneous injection, varying from fibromata to highly malignant sarcomata.—Pulmonary Tuberculosis with the Bovine Type of Bacillus in the Sputum: Fourteen New English Cases, by W. M. Cumming, W. M. Foster, R. O. Girdwood, and A. S. Griffith (Grassington, Bradford, and Cambridge). Ten showed evidence of previous glandular involvement. Two autopsies showed no clear difference from cases due to the human bacillus. From one case bacilli of both types were isolated.—Incidence and Correlation with Clinical Severity of "Gravis," "Mitis," and Intermediate Types of Diphtheria Bacillus in a Series of 500 Cases at Leeds, by J. S. Anderson, K. E. Cooper, F. C. Happold, and J. W. McLeod (Leeds). The differentiation previously announced is held to be confirmed: in 425 gravis cases there were 78 examples of paralysis and 50 deaths; in 113 mitis cases, 2 cases of paralysis and no deaths. The distinction between the two in animal experimentation is not so clear.—A Case of Circumscribed Lymphangeiomatous Dilatation of the Lingual Papillæ, by S. McDonald, Junr. (Newcastle). In a boy of 5.—A Method of Staining the Elementary (Paschen) Bodies of Vaccinia, by J. Craigie (Toronto). Mercurochrome, methylene-blue, and methylene azure.—The Influence of Purified Testicular Extract upon the Rate of Absorption of Antitoxin, by D. McClear and W. T. J. Morgan (Elstree). The extract accelerates the passage of antitoxin into the blood after subcutaneous but not after intramuscular injection.—Conditions Predisposing to Urinary Infection after Post-operative Retention, by C. E. Dukes (London). The longer there is any stagnant urine in the bladder, the more likely it is that the few organisms which are always present will be able to gain an infecting foothold.—Phases in the Growth of the "Virus" of Pleuropneumonia on Artificial Media, by J. C. G. Ledingham (London). Minute spores give rise to a delicate mycelium on which fruiting bodies develop from which a variety of bizarre forms arise; subsequently involution sets in and the spores alone remain to start a fresh cycle.

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MEMORIAL TO THE MAYORESS OF TORQUAY.—It is proposed to provide an X ray apparatus and an operating table at Whitecliffe Tuberculosis Hospital as a memorial to Mrs. Thomas, the mayoress of Torquay, who was recently killed in a motoring accident.

THE LANCET

*LONDON: SATURDAY, FEBRUARY 4, 1933*

## THE EPIDEMIOLOGY OF WHOOPING-COUGH

In a paper [1] already reviewed in these columns, Dr. PERCY STOCKS brought out the fact—still too little appreciated—that whooping-cough is now one of the major causes of death in early childhood. In the sequel concluded on p. 265 he shows that in London it must indeed be regarded as the most expensive epidemic disease among children—that is, when the cost is measured in terms of loss of life, acuteness of physical suffering, permanent damage to respiratory, cardiac, or nervous mechanism, and loss of school attendance. Probably the tendency to under-rate its importance, that is yet apparent in the lay and medical mind, is a relic of the latter half of the nineteenth century when its contribution to mortality was overshadowed by those of scarlet fever and diphtheria, both of which caused many more deaths then than they do now. It is partly this attitude to the disease, partly its relative neglect by research workers, that have, no doubt, led to our somewhat scanty knowledge of it and of its control. For instance, in the present state of knowledge Dr. STOCKS believes that the practice of excluding from already infected schools home contacts to whooping-cough cases may actually be harmful. The risk lies in these excluded children spending their time playing with children of pre-school age, for it is on these latter that the mortality falls heaviest. Of the 44,000 deaths in England and Wales attributed to whooping-cough during 1921–30, 97 per cent. were of children under five years of age, while according to Dr. STOCKS's figures for the borough of Battersea the fatality-rate fell from 4 per 100 cases at age 0–1 to 0·2 per 100 at ages 5–10. If exclusion from school is to be of benefit it must be accompanied by effective isolation from younger children, a difficult thing to achieve in poor and overcrowded areas. The contention that "it is best to get it over while they are young" has no justification; for besides the rapid diminution in fatality-rate there is a reasonable chance, of the order of two in five, Dr. STOCKS calculates, that if a child escapes immediate attack he or she may escape the disease altogether.

One of the peculiarities of the epidemiology of whooping-cough that has long been observed is its heavier incidence of mortality upon the female sex. It is the only important cause of death below the age of five years that reveals this peculiarity. Respiratory causes of death, such as tuberculosis, pneumonia, and bronchitis, all show a lower death-rate among girls—15 to 20 per cent. lower than among boys. In measles, scarlet fever, and diphtheria, girls show a slight advantage, about 5 per cent.; yet from whooping-cough they show an extremely consistent disadvantage, their death-rate at ages under five being 20 to 25 per cent. in excess of boys'. From time to time various explanations of this paradox have been advanced—e.g., sexual differences in the formation and development of the larynx, differences in sensitiveness of the nervous system—but no one of them seems adequate to fit the observed facts at different ages. Owing to lack of efficient notification it is not even clear whether this higher mortality-rate denotes a higher incidence of attack amongst females or a higher fatality-rate amongst the attacked (or a conjunction of both). According to the figures which Dr. STOCKS gives for London boroughs there is a significantly greater susceptibility of female infants to attack from birth, and this increases from the fourth year onwards to a 50 per cent. excess by the tenth year. He has no figures to show whether they succumb more easily to attack, but finds in Greenwich that this female susceptibility bears some relation to season. At ages under seven there was in this borough no excess of girls amongst cases commencing from October to January, but an excess of about 20 per cent. amongst cases arising during the other months.

Another curious point brought out by these figures is that the proportions of children known to their parents to have had whooping-cough before entry to school at ages 3, 4, 5, and 6 are greater in those boroughs of London where the population is less dense and better housed. Though, obviously, this may be partly accounted for by more accurate observation and diagnosis in such areas, Dr. STOCKS believes this factor is insufficient explanation, and that increased crowding of the population in the different districts of London not only does not result in increased incidence of whooping-cough but rather the reverse. He believes that the phenomenon can only be explained in terms of latent immunisation. A possibly related phenomenon has been repeatedly drawn attention to by the Registrar-General of England and Wales in his recent annual reports. In this country the mortality from whooping-cough is invariably lower in the rural districts, and yet with decreasing urbanisation a higher proportion of the deaths fall in the first year of life. For instance, in 1930, of the whooping-cough deaths at all ages in rural districts 53 per cent. were of infants under one year old; in the county boroughs the percentage was only 46. In many years still larger differences are to be found, and the characteristic can be observed at least three-quarters of a century ago, when the death-rate from whooping-cough was three times as high. Measles, on the other hand, shows rather what would be expected—the more the overcrowding the earlier the chance of attack and death. Without more knowledge of the case-incidence of the disease in various areas—to which the present study materially contributes—it seems impossible to explain this further curiosity of a disease still epidemiologically obscure.

[1] Jour. of Hyg., 1932, xxxii., 581; see THE LANCET, 1932, ii., 1115.

## THE REPAIR OF LIVER INJURY

No organ in the body is more competent than the liver to restore itself to normal by reparative and compensatory growth after certain types of injury. Complete removal of one lobe is followed quickly by compensatory hypertrophy of the remaining parts, and in this way a loss of as much as two-thirds can be made good. Similarly the atrophy produced by tying a part of the portal vein is balanced quantitatively by overgrowth of the healthy parts. When, too, the central part of each lobule throughout the whole liver is destroyed, as in chloroform poisoning, regeneration soon reproduces a normal liver, and this takes place even in starving or ill-fed animals in which the necessary materials for reconstruction are drawn from other tissues rather than from the food.

In all these instances, restoration appears to be perfect, permanent, and functionally quite adequate. But there are other types of injury which seem to be more than the liver can deal with at present, though we may suppose that under the sifting influence of evolution it may some time acquire the faculty of coping with these also. A good example is given by Dr. A. BOLLIGER and Dr. K. INGLIS [1] of Sydney, who have studied the action of X rays on the liver. A single massive dose applied to the exposed organ causes a varying amount of acute necrosis of the liver cells, mostly towards the surface but to some extent also in the depths. This may have a lobular distribution, in which case as usual the central cells are most vulnerable, or it may be massive—i.e., without any reference to lobules. If it is extensive enough, the animals die. If they survive the immediate damage, the dogs seem quite well, and biopsies may show little anatomical change. But sooner or later—5 to 16 months in the experiments described—the animals begin to wilt, lose weight, develop jaundice and ascites, and die with the rather vague symptoms of cholæmia. After death the liver is small and irregularly cirrhotic, with perhaps some areas of indefinite hypertrophy: microscopically there is much overgrowth of connective tissue and disappearance of liver cells, and, in addition, areas of recent necrosis are also generally found. The injury inflicted by X rays is evidently one from which satisfactory recovery is difficult or impossible. The anatomy, histology, and clinical history are reminiscent of the condition in man often called "subacute atrophy," many cases of which have been due to poisoning with trinitrotoluene or organic arsenicals. In this the liver cells have certainly not lost their capacity for responsive growth, for nodular masses of hypertrophy are part of the characteristic picture. But hypertrophy or regeneration which will be functionally adequate seems to be, often at least, impossible, and after a long illness the patient dies of liver insufficiency.

Much the same difficulty arises in trying to explain exactly what happens in ordinary cirrhosis. Often enough the individual lobules are considerably hypertrophied, and larger masses of fresh-grown liver cells are frequently present, yet the disease goes on to an inevitably fatal end. What is the essential difference between chloroform necrosis, which cures itself, and these other forms, which do not? It seems as if it must depend not so much on the destruction of the liver cells themselves, but on the involvement of the framework in which they live, and especially perhaps of the finer details of the complicated blood-supply. In the portal canals the hepatic artery breaks up into capillaries which reunite into venules which join the portal venules to enter the sinusoids at the periphery of the lobules. What this arrangement means functionally we do not yet know. Pathologists, too, have many reasons for thinking that the several concentric zones into which the cells of a lobule may be divided probably have different functions, the due discharge of which may well depend on each of them receiving the right sort of blood. This presumptive guess that the important error in X ray necrosis, in T.N.T. poisoning, and in cirrhosis, is a disorganisation of the blood-supply is borne out by the work of A. H. MCINDOE,[2] who showed, by some beautiful injections, how the normal arrangement is completely upset in ordinary cirrhosis with the result that the liver cells receive relatively little of the portal supply. Incidentally, the facts make it difficult to believe that cirrhosis begins with any necrosis which is strictly lobular in distribution.

[1] Jour. Path. and Bact., January, 1933, p. 19.

## BIRTH CONTROL METHODS AND RESULTS

IN this country the birth control movement has now passed out of the crusading phase into the phase in which it has become self-critical and analytical. One of the first signs of this new attitude was the appearance in 1930 of Mrs. SECOR FLORENCE'S "Birth Control on Trial," [3] in which were summarised the results of a follow-up investigation conducted by her upon the first 300 women who attended the irth control centre in Cambridge. A similar critical attitude characterises the successive publications of the International Medical Group for the Investigation of Contraception which have been reviewed from time to time in these columns. ENID CHARLES [4] has now provided a valuable addition to this class of literature.

It is by no means easy to form an impartial estimate of the results of the practice of contraception. Hitherto practically all the published findings have been based upon the records of birth control clinics—that is to say, of special centres, for the most part organised and financed as private enterprises, by groups of interested persons throughout the country. These findings are open to the criticism that they are based upon the experiences of a selected group of women. A woman may attend a birth control clinic for several reasons, among them because a method previously employed has failed. The clientèle of the clinics cannot be taken as representing unselected examples of married women in the community. In order to meet this criticism, the

[2] Archives of Pathology, 1928, v., 23.
[3] See THE LANCET, 1930, i., 919.
[4] The Practice of Birth Control. By Enid Charles, M.A., Ph.D. London: Williams and Norgate, Ltd. 1932. Pp. 190. 10s. 6d.

statistics subcommittee of the Birth Control Investigation Committee drew up, four years ago, a questionnaire which was sent out to women who, so far as possible, were unselected and therefore represented a random sample of the population. It was filled in by the women themselves who were asked not to reveal their names. The material thus furnished provided evidence of a different nature from that yielded by the records of the birth control centres which are compiled by the lay workers and doctors attached to the centres. The questionnaire sought to obtain unbiased answers on the extent to which birth control methods are used in the community, and on the reliability, practicability, effects on health and effects on fertility of different methods. Unfortunately it proved difficult to secure the coöperation of women of the working classes, and the questionnaires were for the most part filled in by women falling in Group I. of the Registrar-General's report of the 1911 census. The greater part of ENID CHARLES'S book is devoted to an analysis of these questionnaires. It also contains analyses of 198 questionnaires sent out by a nurse to women upon whom, in the years since the war, she had fitted Dutch pessaries, and of 400 follow-up reports carried out by the Birmingham Women's Welfare Centre. The volume therefore contains an abundance of statistical material, and, by reason of the fact that the generalisations it contains are based upon first-hand information, stands out from a mass of propagandist literature founded on little else than moral preconceptions and biased impressions. Though it cannot be claimed that any of the three sources of material represents a random sample of the population, they furnish, when taken together, a valuable body of evidence from which it will only be possible to summarise here the salient facts of medical interest.

Of 432 women, then, who vouchsafed their experiences, only 12 had employed no contraceptive methods whatever. It will thus be clear that in the group investigated, in which the active reproductive life averaged 12 years, birth control methods of one sort or another are very extensively used. ENID CHARLES concludes that the sheath and the rubber cap are the most reliable methods, provided that they are used in conjunction with a spermicide or a douche. In these circumstances, assuming that coitus takes place about once a week, the odds against a single conception occurring over a period of about five years are about 17 to 1; and the odds against conception following a single intercourse are about 5000 to 1. Of the two methods above referred to, the combination of a sheath and another method gives the highest degree of reliability, the Investigation Committee's questionnaires here showing a percentage of success of 98 comparing with one of about 75 for the rubber cap combination. A soluble quinine pessary, when used alone, was found to be relatively unreliable, the percentage of success being only 54. The rubber cap combination seems to be somewhat more acceptable on æsthetic grounds than the sheath, which is disliked by a number of the husbands. A question upon the effects of the use of birth control on health and conjugal well-being was answered by 339 women. Of these, 2 found contraceptive methods so objectionable that they were given up, 37 found them unsatisfactory but inevitable, 193 found them in general terms satisfactory, and 107 spoke of them as essential to health and marital happiness; but there was evidence that coitus interruptus was found harmful by many couples who, at one time or another, had recourse to this method. The Investigation Committee's questionnaires also provided evidence on the effects of contraception on fertility. In a series of 420 women desiring pregnancy there were only 17 who were unable to conceive. Allowing for the fact that the sample may be selected as regards absence of sterility, this figure compares favourably with the amount of involuntary sterility which has been computed to exist in the general community, wherein one-sixth of all marriages are thought to be sterile. But ENID CHARLES adduces evidence that despite its unreliability as a contraceptive, quinine may have an effect on fertility in that, for users of pessaries containing this substance, the period intervening between the discontinuation of use and the moment of conception seems to be unduly prolonged. These are but a few of the findings set forth in this book which should be read by all persons seriously interested in the sociological and clinical aspects of contraception. She has analysed and summarised the complicated material placed in her hands with deftness and lucidity; and the caution with which she advances her conclusions should be an example to all writers upon the subject.

Several of these conclusions are borne out by the annual report for 1931–32 of the Society for the Provision of Birth Control Clinics.[5] Of 97 women who discontinued practising birth control because they wanted a child, 96 became pregnant; and 158 women who, on one occasion, neglected to use the method advised, had unwanted pregnancies. The report further confirms ENID CHARLES's findings about the frequency with which coitus interruptus is practised by working-class couples, and also about its unreliability. The medical officers of the Walworth women's welfare centre also draw attention to the frequency with which prolonged lactation fails as a contraceptive measure. The method most frequently advised at this centre is a combination of Dutch pessary, a spermicidal ointment, and a douche of soapy water. A case is mentioned of a woman who used the same pessary and syringe for five years. An increasing number of couples are successfully using the sheath. A list of the 17 subsidiary clinics affiliated to the society is available to practitioners. At these clinics up to August 31st, 1932, advice has been given to 47,500 women. Printed leaflets showing the places where centres affiliated to the society have been established, and the times at which sessions are held at these centres, are available on demand.

[5] Obtainable from the Secretary, 153A, East-street, Walworth-road, London, S.E.17.

# ANNOTATIONS

## EARLY DIAGNOSIS OF MALIGNANT DISEASE

LESS than a year ago [1] we chronicled the formation of the Harley Medical Society, which originated in a desire to foster the scientific spirit among members of the medical group attached to the London Clinic and Nursing Home. Mr. Lockhart-Mummery gave the opening address entitled the Origin of Tumours. We have now received (see p. 254) a report of the inauguration of yet another more specialised society, the London Cancer Society, which first met last week, under the presidency of Mr. Lockhart-Mummery, at 144, Harley-street. It seems doubtful whether the multiplication of more societies in an area already well served by the different sections of the Royal Society of Medicine and by the Medical Society of London will really lead to the advances in knowledge anticipated by some of its members. The proposition put forward at the meeting was that, as regards diagnosis at any rate, knowledge had reached the stage where, if the public would only consent to submit to periodical examination, and all available accessory methods of diagnosis were enlisted, very few malignant growths would remain undetected at an early stage. If we cannot share this optimism it is perhaps a matter of definition. Evidently "an early stage" is considered to be a stage at which the growth though visible or palpable is still operable and no metastases have developed. But every practitioner could reveal tragic stories of cases where vigilance has failed to detect the beginning of malignancy. In fact, except by accident, for example during the routine microscopical investigation of a uterus or a breast removed for another reason, it is probable that really early lesions are never detected. By the time they are palpable or visible by X rays or to the naked eye, they may well, in certain regions, be inoperable. And in the intervals between any routine periodical examinations—which, to be in any degree reliable, must include examination per vaginam and per rectum—a quickly growing tumour may defeat the whole elaborate scheme. Whether the number of growths detected in this way is likely to be worth the risk of developing in healthy persons morbid fears of disease, or of increasing such fears in those who already suffer from them, is a matter of opinion. Mr. Lionel Colledge, in the current issue of the *Practitioner*, which is devoted to articles on the early diagnosis of malignant diseases, says wisely that "it is the doctor, and not the patient who should be the cancerphobe." It is the doctor, too, who will get some surprises from a study of these 14 articles on diagnostic possibilities in cancer of the tongue, the stomach, the throat, the mediastinum, lung, and pleura, the colon, the rectum, the breast, the uterus, bone, the bladder, the prostate, and the testicle. For example, while Mr. Geoffrey Keynes admits that early cancer of the breast is not easy to diagnose, Dr. Izod Bennett boldly declares in connexion with the early diagnosis of carcinoma of the stomach, that "in any particular individual the question of whether or not cancer of the stomach is present can be answered accurately in 99 out of every 100 cases." Obviously the definition of "early" as used by different observers can bear no firm relation to ultimate results of treatment, since the prognosis in breast cancer is immeasurably better than in cancer of the stomach. The omission from the series of the brain and the kidney regions, where diagnosis is especially difficult, is regrettable. The articles are all provocative. Sir Holburt Waring contributes a general introduction in which he outlines the methods of examination available and utters some useful warnings. The conclusion is inevitable from the careful study of these articles that in spite of the many instruments and direct and indirect methods of examination now available, the early diagnosis of malignant diseases is still often very difficult.

[1] THE LANCET, 1932, i., 619.

## YELLOW FEVER IN FRENCH WEST AFRICA

IN the English territories of Nigeria yellow fever is endemic in the coastal regions. This endemicity decreases as one proceeds into the interior, and in the southern zone the disease can only be recognised in its epidemic form. This knowledge is the outcome of studies carried out at Lagos, under the auspices of the Rockefeller Foundation of New York, by the West African Yellow Fever Commission, whose director, Dr. Beeuwkes, has since been pursuing his investigations further north in the French territory by permission of the Governor-General. His object was to try to discover any manifestations there of yellow fever, and to ascertain the most northerly limit of this terrible disease. Dr. Beeuwkes has had the collaboration of Dr. C. Durieux, who joined the members of the Commission in Lagos in April last, and now describes the work undertaken and in progress.[1] The members of the Commission detailed to undertake the expedition included Dr. Mahaffy as leader, Mr. Michailoff as secretary, and various native personnel. They left Lagos on April 11th by rail for Kano, whence further journeys were undertaken by motor-car. On April 14th they reached Zinder, and passed through various townships of the French Niger Colony, the French Sudan, and Dahomey. During the journey 803 specimens of blood were taken in different localities. Wherever possible the data were derived from natives born and bred in the neighbourhood. The determination of the exact district to which different natives of Nigeria belong is extremely difficult on account of the shifting nature of the population; specimens were obtained from women by preference, as they seemed less mobile than the men. The specimens obtained were tested according to the method of sero-protection used by the American Yellow Fever Commission at Lagos. Sawyer's method of sero-protection is the one favoured; a starchy emulsion is first injected into the brain of a white mouse, and two hours afterwards a mixture of serum-virus is injected into the peritoneal cavity. Each serum is injected into six mice, and the different operations are carried out as speedily as possible; 25–30 sera are tried out every three days, and about 300 sera a month are thus tested. The white mouse apparently is the ideal animal for testing susceptibility to the yellow fever virus; up to the present no less than 2000 sera from all parts of Nigeria have been tested. The results of the tests carried out with the specimens of blood collected on the tour of the French territories did not favour the general opinion that the French Niger Province has been free from yellow fever up to recent times. These tests indicated that the disease has existed for several years in the population of this colony. In the Zinder region, the last manifestations of yellow fever appear to have been between the years 1915 and 1919 (the youngest of the positive

[1] Annales de l'Institut Pasteur. December, 1932, p. 735.

cases were about 17 years old). At present the disease is localised in the southern zone of the colony on the road from Gouré to Niamey, and it has not yet spread northwards. Dr. Durieux expresses the hope that these investigations will be pursued in order to define the areas where yellow fever is endemic, so that prophylactic measures can be undertaken, and the areas as yet free from the disease protected from invasion.

## SEX HORMONES AND UTERINE INFECTION

IT is well known that the reticulo-endothelial system plays an important part in resistance against infection, and a fair amount of evidence has accumulated to show that any factor which stimulates the local reaction of reticulo-endothelial cells is of importance in combating a local infection. The reaction of the reticulo-endothelial system in cases of puerperal sepsis was investigated some years ago by Louros, who pointed out that the utility of such methods of treatment as the injection of foreign proteins, of colloidal substances, and of milk probably depends on their stimulating the reticulo-endothelial system. C. F. Fluhmann [1] has lately described experiments on the rabbit uterus which were designed to show the local response of reticulo-endothelial cells to the injection of ovarian and pituitary hormones. The usual technique of injecting trypan-blue was employed, and the test animals were spayed rabbits and rabbits under the influence of œstrin and of pituitary hormones. The local response in the uterus was either one of œstral or progestational hypertrophy. The results obtained showed that either under the influence of œstrin or when in a state of progestational proliferation the uterus contained a large number of macrophages, and it was found that if local injury to the uterus was previously induced by inserting a silk ligature into one of the uterine horns the macrophage response was tremendously increased. Fluhmann tries to make out a case for treating puerperal sepsis and pelvic inflammations by the administration of the various ovarian and pituitary hormones. It is, however, fairly clear that the work is only preliminary, and it is quite likely that other interpretations may be given to his results.

## RECENT WORK IN HÆMATOLOGY

THE method adopted throughout the most recent volume [2] of the Practical Medicine series devoted to General Medicine is to abstract the literature of the last 18 months dealing with each subject discussed. The abstracts are clearly presented, but little attempt at criticism is made, nor is a complete picture of any disease or its treatment given. While this method has obvious disadvantages from the point of view of the student, it results in an extremely valuable book of reference for workers in specialised fields, since papers from less well-known journals in many languages are included in the survey. In the section on diseases of the blood, which has acquired much clinical importance in the period under review, Dr. George R. Minot and Dr. W. B. Castle cover the whole field of hæmatology. The papers included deal, as the editors say, "with fundamental physiological questions or techniques of importance in normal and pathological conditions of the blood." The treatment of polycythæmia vera with potassium arsenate is discussed. Cases which have been followed for several years, and unsuccessfully treated with both phenylhydrazine and X rays over the long bones, have shown prompt clinical and hæmatological improvement on administration of potassium arsenate. Remissions have been maintained by continuous treatment, and no untoward effects are recorded. Attention is drawn to the striking clinical parallelism which exists between exophthalmic goitre and chronic lymphatic leukæmia. It is suggested that the beneficial result of treatment with Lugol's iodine in both conditions is due to its action on the sympathetic nervous system. An explanation of the failure of complete gastrectomy in dogs to produce the blood picture of pernicious anæmia is discussed. It has recently been shown by clinical trial that the dog's stomach normally lacks Castle's intrinsic factor. Therefore removal of the stomach produces no deficiency of this factor, and the blood picture of pernicious anæmia, due to such a deficiency in man, does not result. Experiments are described which suggest that human liver acts as a storage for the principle effective in pernicious anæmia. Comparison of the effect of an extract prepared from the liver of a patient with pernicious anæmia who died without treatment, with that of an extract from the liver of a patient dying during a remission, showed the latter to be potent while the former was inactive. The full indices of authors quoted and of subject matter will be appreciated by the research worker in this rapidly growing subject.

[1] Amer. Jour. Obst. and Gyn., 1932, xxiv., 654.

[2] General Medicine. Practical Medicine Series, 1932. Edited by George W. Weaver, M.D., Lawrason Brown, M.D., George R. Minot, M.D., William B. Castle, M.D., William D. Stroud, M.D., Ralph C. Brown, M.D. Chicago: The Year Book Publishers. Series 1932. Pp. 837. $3.

## PREVENTION OF NERVOUS DISORDER IN CHILDHOOD

CONDITIONS of modern civilisation have made the "nervous child" a figure unfortunately only too familiar. Although it is difficult to estimate with any degree of accuracy the incidence of what has variously been called nervous disorder, nervous instability, or neurosis in any age-group, there is little doubt that it has increased in the last 30 or 40 years, particularly in children of school age. The proceedings of the regular winter conference of educational associations (some sessions of which were reported in THE LANCET of Jan. 14th, p. 102) are sufficient testimony to this increase. Many of the operative factors are beyond medical control. The perpetual noise and speed of modern traffic, the increasing number of "one-child" marriages, and the economic difficulties of providing the town child with adequate outdoor playgrounds, are examples. Other environmental factors can be modified by a more enlightened system of education in the home and school. The prevention of nervous disorder in childhood formed a main subject of discussion at the second meeting, held in Geneva last September, of the *Association Internationale de Pédiatrie Préventive*. Whatever part heredity, innate disposition, or so-called organic disease may play in the individual case, it was generally conceded that environment and education will always influence the condition either for good or for bad. There is in fact no such things as a pure endogenous or pure exogenous neurosis. Attention was directed almost exclusively to a consideration of the extrinsic influences brought to bear on the individual child. Prof. F. Hamburger emphasised the importance of an artificial manner of life in the production of neurosis; not only is the child liable to receive inadequate exercise, inadequate exposure to light and air, and an unnatural dietary, but the modern parent is prone to focus a quite unnatural

degree of attention on such activities as feeding, excretion, and cleanliness. Fussiness over functions which should normally be largely automatic can only serve to produce a state of unrest in the child's mind, and may result in the very symptoms it is desired to avoid—anorexia, constipation, enuresis. The first essentials of the parents' attitude should be kindness and consistency, with willingness to let the child learn as far as possible from his own experience without unnecessary interference. Several speakers considered the importance of place in family in influencing development; it was pointed out that while a first child may find it intensely wounding to be superseded by a new baby, the psychological trauma is ultimately far less damaging than retaining the position of only child indefinitely. Dr. Heinrich Keller, in a recent study, has similarly observed [1] that the nervous child in Vienna is frequently an only child, and is characterised by an unsocial rather than an antisocial attitude to his contemporaries—lack of desire to make friends from a feeling of superiority. He gives as an example the reply of a 10-year-old boy, the only son of a widow, when asked if he had many friends in the school: "Nein. Ich bin doch ein feines Kind and das sind lauter so Buben!"

In England a subcommittee of the National Council for Mental Hygiene, under the chairmanship of Dr. H. Crichton Miller, recently attempted to state [2] approximately the demand made upon children of various ages, and to indicate the nature of the individual response, the material for the study being obtained from the answers to a questionnaire issued to the heads of 39 schools of all types, with a total attendance of over 10,000 children between the ages of 2 and 18 years. Whilst an investigation of this kind should prove an invaluable preliminary to the removal of any unnecessary stress upon the growing child, it is clear that much of the anxiety and strain of school life is directly traceable to the economic pressure which demands examination qualifications, regardless of the best interests of physical and mental health. Under the present examination system a time factor is introduced and a definite result (which may be of great importance to the child's after-life) is often required by a certain date; the backward child is pushed beyond his capacity, and the precocious child urged to further efforts. Even in schools which pride themselves on scholars taking examinations in their stride, it is pointed out that the length of the stride may be profoundly modified. The two periods which appear to be associated with a maximum of nervous instability and a maximum of punishment—namely, puberty (13–14) and during adolescence (16–17)—are both preceded by a period of working for examinations. Crowding of the time-table also tends to result either in physical education being neglected, or (in schools where games are taken with great seriousness) in rest-periods being quite inadequate. Emphasis is laid on the value and importance of a period of rest in the horizontal position for examination candidates as much as for younger children, though the time devoted to this each day was practically nil in all the time-tables of the 14–18-year-old groups. Indeed, although State infant schools (2–8 years) are the best provided in this respect, in the primary (8–14) group no such provision is made in any of the schools under consideration. For adolescent girls there is a convention which permits intermittent relaxation at school; for boys there is none. The effects of prolonged fatigue upon nervous stability, the report remarks, cannot but be harmful. The report also draws attention to the value of training in accurate movement and performance in inducing nervous stability, and the need not only for the inclusion of handicrafts and gymnastics in the time-table, but for properly trained teachers in charge of these subjects. The report concludes with the suggestion that further research might profitably be directed towards the following problems of school life: size of classes, the number of staff teaching any one child, the hours of sleep and daily rest, recording of height and weight, study of movement as an index of vitality, creative effort, examinations, terminal strains (especially about the eighth week), and punishments.

[1] Wien. Klin. Woch., 1932, xlv., 1180.

[2] School Life and Nervous Instability. With a foreword by Sir Maurice Craig. Nat. Council for Mental Hygiene. See THE LANCET, 1932, ii., 354.

## ANÆSTHESIA IN ANIMALS

ALTHOUGH there are as yet no acknowledged specialists in anæsthesia for veterinary work, yet the knowledge of the science and the technique of administration for animals do not lag behind those employed with human patients. Naturally there are differences in degree and this became particularly evident in Prof. Wright's account of nembutal narcosis in the dog and cat in the course of the discussion at the section of comparative medicine of the Royal Society of Medicine on Jan. 25th. The veterinary surgeon uses nembutal in doses and for a purpose which are not, as Dr. I. W. Magill showed, desirable in the human subject. Nembutal appears to be a good and safe anæsthetic for the lower animals. For human beings its use is mostly restricted to that of an excellent paver of the way for true anæsthesia. One speaker related a case illustrating the extraordinary capacity of dogs to withstand the action of this drug. A dog of 20 lb. in weight received, for lethal purposes, 10 capsules of grs. 1½ nembutal. Next morning the dog was still comfortably alive, although with most reflexes in abeyance, and prussic acid had to be employed to ensure death. The usual dose of nembutal employed for animals appears to be up to gr. ¼ per lb. of body-weight and the results highly satisfactory, no additional anæsthetic being required in many instances. Two papers dealt with epidural analgesia and with anæsthesia for thoracic operations. With regard to the former, opinion was expressed that the method was invaluable for cattle and not sufficiently employed in this country.

Mr. L. O'Shaughnessy, who gave an admirable account of the subject, recommended positive pressure anæsthesia by means of a mask for operations within the chest. He demonstrated the Tigel-Henle apparatus adapted for use in animals and contended that with full narcosis there was no risk of laryngeal spasm, and therefore no occasion for endotracheal anæsthesia. Dr. C. F. Hadfield described a successful anæsthetic given by him for tooth extraction to a 20-year-old Pekinese.

## TRENCH FEVER UNDER PEACE CONDITIONS?

A DISEASE of war, springing up mysteriously in 1915 and disappearing as mysteriously in 1919—such is the generally accepted view of trench fever, now considered of historical interest only. Yet no one has adequately explained its complete disappearance after the late war, although its vector, the louse, has survived the conflict. Dr. P.-J. Braslawsky,[1] of Kieff, who reports a case much resembling trench fever, says

[1] Paris méd., Jan. 14th, p. 41.

that during the typhus epidemic of 1920–21 there was a considerable number of cases in the neighbourhood of Kieff of which he personally observed 22. Since then he has seen from 1 to 3 cases yearly, up to 1925, making 32 cases in all. He suggests also that the Wallachian or Moldavian fever described by Dehio in the Russo-Turkish War of 1877–78 was identical with trench fever, and that a series of these cases among the children of a Berlin family described by Apolant in 1904, as well as that of a nurse recorded by Weitz in 1910, were also examples of the disease. Further, a five-day fever, endemic in Galicia for a long time before the war, has been identified with trench fever by Tchirikov and by Francke. Braslawsky therefore thinks it reasonable to suppose that trench or (as he prefers to call it) five-day fever is endemic in certain parts of Eastern Europe, appearing in occasional and often unrecognised cases, and forming a reservoir in the civilian population from which great epidemics may originate in times of war. The only other cases reported since the end of the war appear to be two in Japan recorded by F. Katsurada and J. Yoshino in 1927 [2]; but rare diseases are seldom noted unless they are looked for. Although the one recorded by Braslawsky showed four characteristic rises of temperature about every fifth day, it was not typical, since the tenderness of the tibiæ recorded by various observers in proportions of from 50 to 80 per cent. of cases was not present. Unfortunately the only pathological investigation carried out was to exclude the presence of malarial parasites. Presumably the best proof of the continued existence of the disease would be the agglutination of rickettsia bodies from the excreta of lice fed on suspected trench fever patients in the presence of immune serum from animals immunised with infective lice excreta, on the lines of the work of J. C. G. Ledingham.[3] Since the blood of infected persons has been found to be still infective for a period up to 443 days [4] such examination should not prove impossible.

## REPORTS ON CHILD GUIDANCE

As the outcome of a White House Conference on Child Health and Protection, called by President Hoover, a subcommittee on psychology and psychiatry was appointed, consisting of eight distinguished psychiatrists, including Prof. Adolf Meyer. Their report, now issued,[5] may be accepted as the considered view of those in the United States who have had most experience of child guidance. Since it is in that country that there has been most activity in this field, and perhaps also most enthusiasm and most optimism, it is of special advantage to get so sober and moderate a review of the subject as is to be found here. Propagandist exaggerations of the need of psychiatric treatment for children, and of the advisability of prompt interference are not accepted, nor the extravagant claims for personnel and time in running such clinics. The place of the general practitioner, the pædiatrician, the social worker, and others who may be concerned is considered temperately. An account of the discussions of the subcommittee is included, and an appendix, with sections on clinics with definite relation to courts, and on special training centres and schools such as the Child Guidance Institute in New York and the Merrill-Palmer school. A valuable section on psychiatric social workers, written by Miss Mildred Scoville of the Commonwealth Fund, provides a clear and vigorous contribution to the difficult question of the relation of these indispensable workers to the psychiatrist and to general social and medical problems of adjustment. This volume is by far the most useful survey of the broader aspects of child psychiatry that has as yet appeared, and should be studied by all who are interested in the development of the work in this country.

The British Child Guidance Council, subsidised by the Commonwealth Fund, has now been established for six years, and has instituted in London, Glasgow, Liverpool, Manchester, and Birmingham clinics for treatment and for training social workers in the delicate task of ascertaining how the child reacts to the environment of home and school, and of altering that environment when necessary. Since the child's environment largely consists of its parents, any attempt to alter it means a systematic education of the parents, and so the child guidance worker is concerned more directly with adults than with children. Dr. William Moodie, at a meeting of the Council held on Jan. 23rd, pointed out that besides instructing its workers the Council undertakes widespread propaganda directed to teachers, doctors, and social workers in other branches. Dr. R. H. Crowley laid great stress on the advice given in the last annual report to child guidance workers: to be sensitive to changing needs and equipped to meet them.

## PNEUMATIC MACHINERY AND JOINT LESIONS

EVERY step in industrial progress is apt to bring with it some unforeseen addition to the roll of occupational diseases. Recent reports suggest that machinery driven by compressed air is not guiltless in this respect. Pathological changes in various parts of the body have been described in workers handling pneumatic drills, notably lesions of the bones and joints of the upper limb. The condition has apparently attracted little attention in England, but in Germany especially it is a hazard scheduled under the Workmen's Compensation Act as an industrial disease. This official recognition has been criticised on the ground that the clinical picture is still somewhat vague, and the ætiological relationship with pneumatic drills not established beyond doubt. In a recent paper [1] Meiss sets forth the results of an attempt, to end this uncertainty. His conclusions are based on an examination of 107 men employed as riveters or assistants. As a control, 80 manual workers from other fields of labour and 30 non-manual workers were examined. In the first group, 24 men showed changes in and around one or both elbow-joints, and in several cases other joints of the upper limb were also affected. It is suggestive that in the only left-handed worker the condition affected the left elbow only, while in the remainder the right arm was the more markedly involved. In the affected elbow-joint the appearances revealed by X rays resembled those of osteo-arthritis, more especially of the head of the radius. Osteo-arthritis of the elbow-joint was found in five of the larger and one of the smaller control groups, and thus cannot be regarded as necessarily due to the use of pneumatic tools. Of greater diagnostic import, in Meiss's opinion, is ossification in the origin of brachialis anticus, forming a spur upon the front of the humerus just above the elbow. This was found in 20 cases, and in none of the controls with the exception of one man

[2] Arbeiten u. Tropenkrankh. Festschrift for B. Nocht., 1927, p. 225.
[3] THE LANCET, 1920, i., 1264. [4] Jour. Hyg., 1921, xx., 258.
[5] Psychology and Psychiatry in Pediatrics: The Problem. Report of the Subcommittee on Psychology and Psychiatry. Edited by Bronson Crothers, M.D. New York: The Century Co. 1932. Pp. 146. 7s. 6d.

[1] Nederlandsch Tijdschrift voor Geneeskunde, Dec. 31st, 1932, p. 5902.

who was found on inquiry to have formerly worked with a pneumatic machine. In some cases ossification was present in other neighbouring muscular attachments—viz., the insertions of biceps, triceps, and brachialis anticus; as Meiss points out, these are the muscles which fix the elbow-joint, and are necessarily exposed to intermittent increase of tension with each vibration of the machine. In several cases there were arthritic manifestations in the carpo-metacarpal joint of the thumb which Meiss claims to be the first to describe. The thumb is used to grasp the lever controlling the machine, and the joint is thus directly exposed to the vibrations. In one case the right acromio-clavicular joint was affected, in a man who had been accustomed to steady his tool against the right shoulder. Of other conditions ascribed to the use of pneumatic drills, including necrosis of the semilunar bone and arthritis of the sterno-clavicular and shoulder-joints, no examples were found in the present series. The clinical features included pain in the elbow-joint, which is the joint most commonly affected, the pain coming on after ceasing work (the "Ruheschmerz" of German authors), and gradually disappearing when work was resumed; also slight limitation of flexion and extension. There was little or no limitation of pronation and supination; creaking was rarely, slight muscular atrophy frequently, observed. It is emphasised that none of the men had previously complained of the pain, and that there is practically no disability connected with the condition. Where other joints were affected, similar clinical features were noted. Meiss concludes that the evidence points to a probable connexion between joint lesions and the use of pneumatic drills, but that further investigation is required before it can be regarded as proven.

## THE HEALTH OF NEW YORK

THE severe economic distress has begun to manifest itself in New York City, according to a report of the Health Commissioner for 1932, in an increase in the number of new cases of tuberculosis. Unless conditions improve this increase in new cases will, without doubt, shortly be reflected by a rise in the death-rate from phthisis, though in 1932 the death-rate from this cause was actually distinctly lower than in the preceding years. Some of this increase in notified cases may rather be due to the admittedly extra zeal with which the department of health has intensified its work of discovering early cases, and to this extent the evidence against economic conditions is not completely convincing. A further consideration is that both the general (crude) death-rate (10·3 per 1000) and the infant mortality-rate (51 per 1000 live births) reached in 1932 their lowest recorded levels. A cause of death that does suggest a distinct increase in prevalence in recent years is diabetes, the increase being especially pronounced among women over 45 years of age. The New York Academy of Medicine has appointed a special committee, in coöperation with the department of health, to consider this problem. Another similar committee, whose report will shortly be available, has been considering the maternal mortality-rate of the city. The rate registered for 1932 was as high as 6 per 1000 live births.

The cost of the health department to New York was, in 1932, 68 cents per head, of which nearly one-third was devoted to child hygiene activities (covering both pre- and post-natal protection), and one-seventh each to the control of infectious diseases and to the supervision of food and drugs. The responsibilities of the department are wide-ranging, as will be seen from the fact that the statistics of motor-car accidents come under its eye. In New York deaths from motor-car accidents reached a peak in 1929, and in the last three years have shown some decline, which the department of health believes can be credited to the greatly increased number of signal lights installed and the extensive campaign of education in street safety inaugurated by the police department in many of the schools of the city.

## CARBON MONOXIDE FROM PAINT

ATTENTION was drawn not long ago to the danger of carbon monoxide poisoning from the air of sealed compartments on board ship, and it was shown that the gas was derived from the linseed oil of the paint used in these compartments. Further investigations on this subject have been carried out by S. F. Dudley, F. G. Edmed, and R. C. Frederick.[1] The materials used in the investigation were oxide of iron paint, red lead paint, aluminium paint, bituminous compositions, and two grease paints, one of which contained a small amount of linseed oil. These materials were spread inside petrol tins which were then sealed for varying periods. After one, two, or three months' storage the air from the cans was analysed, and its lethal effects estimated by biological experiments on mice. Linseed oil was again found to be the determining factor in the production of carbon monoxide; the paint dries, oxygen is absorbed, and carbon monoxide is evolved. On board ship it seems that after painting and scaling, absorption of oxygen proceeds to such an extent as to produce an irrespirable atmosphere by the time the compartment is reopened. But at an earlier stage of the process sufficient oxygen for respiration may coexist with a dangerous quantity of carbon monoxide. A man entering such an atmosphere might breathe long enough to absorb a lethal dose of carbon monoxide; this, therefore, is a dangerous period. The quantity of paint, or perhaps rather the surface area exposed, as well as the time during which the compartment has remained sealed, will determine whether the atmosphere will poison those entering with carbon monoxide, or "drown" them from lack of oxygen.

WE regret to announce that Prof. William Phillips Graves, the well-known American gynæcologist, died at Boston on Jan. 25th at the age of 63. Professor of gynæcology at Harvard University and surgeon-in-chief to the Boston Free Hospital for Women, Dr. Graves was the author of a text-book on his subject, and was one of the two recipients of the honorary Fellowship at the recent opening of the new house of the British College of Obstetricians and Gynæcologists. Subsequently he attended the tercentenary dinner of the Society of Apothecaries of London, and conveyed a courteous greeting from the branch of the profession which he represented.

## INDEX TO "THE LANCET," VOL. II., 1932

THE Index and Title-page to Vol. II., 1932, which was completed with the issue of Dec. 31st, is now in preparation. A copy will be sent gratis to subscribers on receipt of a post-card addressed to the Manager of THE LANCET, 7, Adam-street, Adelphi, London, W.C.2. Subscribers who have not already indicated their desire to receive Indexes regularly as published should do so now.

[1] Jour. Indust. Hyg., January, 1933, p. 1.

# SPECIAL ARTICLES

## SOME EPIDEMIOLOGICAL FEATURES OF WHOOPING-COUGH

### A STATISTICAL INVESTIGATION

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(*Concluded from p. 216*)

PART II

### Infection within Houses.—Incubation Period

THE principal modes of infection from child to child which can be studied statistically are (*a*) within the same house, (*b*) from house to house, and (*c*) within the same school.

The distributions of intervals between the onset of successive cases within the same house are analysed in Table VIII. for Battersea and Greenwich. Of the 902 intervals of 0–3 days, more than 800 were pairs of cases recorded as starting at the same time, and it may be assumed that in practically all these instances the two children were simultaneously infected from some source outside the house, and did not infect one another. No doubt this also applies to some of the intervals of 4–6 days, of which there were 87 in Battersea and 72 in Greenwich. It is difficult to decide a priori whether it is more probable that the incubation period could be as short as three or four days, or whether the incubation periods of two children simultaneously infected could differ by this amount, or whether there is an appreciable likelihood that two children living in the same house may be infected from the same outside source on different days. This is virtually settled, however, by the form of the curve in Fig. 1 between 0 and 6 days, for this curve must be the resultant of two component curves which cannot differ much from the hypothetical representation of them as dotted lines, and these suggest that direct infections with intervals as short as two days may be a possibility, and also that almost all intervals of four days or more may be attributed to direct infection of the second child by the first.

TABLE VIII

| Interval between onset of cases. | Frequency of intervals. | | | Frequency corrected to a range of one day. | Per cent. of successful plate cultures (Madsen). |
|---|---|---|---|---|---|
| | Battersea. | Greenwich. | Total. | | |
| 0–3 days | 521 | 381 | 902 | ..... | 75 |
| 4–13 „ | 416 | 229 | 645 | 64·5 | 57 |
| 14–20 „ | 178 | 102 | 280 | 40·0 | |
| 21–27 „ | 61 | 58 | 119 | 17·0 | 61 |
| 28 „ | 16 | 6 | 22 | 10·6 | 45 |
| 29–34 „ | 18 | 34 | 52 | | |
| 5 weeks | 18 | 17 | 35 | 2·9 | 40 |
| 6 „ | 4 | 12 | 16 | | 9 |
| 7 „ | 12 | 12 | 24 | 2·4 | — |
| 8 „ | 3 | 6 | 9 | | |
| 9 „ | 2 | 5 | 7 | 1·1 | — |
| 10 „ | 4 | 4 | 8 | | |
| 11 „ | 3 | 6 | 9 | 1·0 | — |
| 12 „ | 4 | 1 | 5 | | |
| 3–6 months | 31 | 38 | 69 | 0·8 | — |

In a large number of instances the interval could be only measured as "one week," an approximate estimate which might mean 6, 7, or 8 days, or even 5 or 9 days. The same difficulty occurs at 14 days. Grouping together 5–9 and 10–17 day intervals in order to escape this difficulty, the Battersea frequencies used in drawing Fig. 1 were as shown in Table IX., and they produce when plotted a perfectly smooth curve. The form of this curve shows that the most frequent interval between successive cases in a house is one week, that the probability of a case occurring at a fortnight's interval is about half as great as this, at three weeks about one-fifth, at a month about one-eighth, and between three and six months about one two-hundredth. It must be concluded that it is possible for the incubation period to be as short even as three days, but it will most probably be a week. The upper limit of the possible incubation period cannot be determined from this data since it is not known how long after onset the infection was conveyed to the second child. Where infection occurs after long intervals the cause is no doubt one of those exacerbations of the more infective catarrhal condition brought about sometimes by superimposed catarrhal infections, which are often noticed in whooping-cough. During such "relapses" to the catarrhal stage it must be presumed that the patient again becomes infective.

### Risk of Family Infection

In the last column of Table VIII. are shown the percentages of successful cultures of *Hæmophilus pertussis* reported by Madsen,[9] as obtained by Danish bacteriologists, when plates of a special medium of potato, horse blood, and agar were held at 6 inches from whooping-cough patients whilst coughing. These percentages were based on 914 cases of whooping-cough tested during the first, second, third, fourth, fifth, and sixth weeks of the disease, and more recently Gardner and Leslie [7] have obtained similar results in England. There is an obvious correspondence between the time required for the two measures of infectiousness to decline to a low level indicative of small risk. Although the practice of terminating isolation at the end of six weeks is justifiable on the grounds of greatly diminished risk, the evidence from plate culture experiments gives no grounds for any dogmatic promise that the danger of infection is entirely at an end, since an infective relapse may occur. The Battersea figures show that in fact one case out of every hundred is followed by another in the same house after an interval between six weeks and six months, a small risk but not entirely negligible.

Selecting any child at random who has developed whooping-cough, what is the probability that another child will develop it in the same house (i) within three days, presumably by having been infected from the same source in most cases, (ii) after three days but

TABLE IX

| Interval, days .. | 0 | 1 | 2 | 3 | 4 | 5–9 | 10–17 | 18–20 | 21–27 | 28–34 | 35–41 | 42–90 | 3–6 mths. |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| Frequency .. .. .. | 465 | 26 | 12 | 18 | 35 | 286 | 231 | 42 | 61 | 34 | 18 | 32 | 31 |
| Mean frequency for a single interval .. .. | 465 | 26 | 12 | 18 | 35 | 57·2 | 28·9 | 14·0 | 8·7 | 4·9 | 2·6 | 1·6 | 0·3 |

within a month by direct infection, and (iii) after a month but within six months? From the Greenwich data the totals at risk to be followed by a second case which could be reported within the range of the records were respectively 4102, 4070, and 3800 for these intervals, and the respective probabilities were therefore 381/4102, 395/4070, and 101/3800 according to Table VIII. For Battersea the totals at risk during 1925–29 to be followed by a second recorded case were in each instance 5574, and the probabilities were therefore 521/5574, 671/5574, and 99/5574. It follows from Table X. that in London about 22

TABLE X

| — | Probability of second case in same house. | |
|---|---|---|
| | Battersea. | Greenwich. |
| Within 3 days .. .. | 0·094 or 1 in 10·7 | 0·093 or 1 in 10·8 |
| 4 to 28 days after .. | 0·120 or 1 in 8·3 | 0·097 or 1 in 10·3 |
| 1 to 6 months „ .. | 0·018 or 1 in 56 | 0·027 or 1 in 38 |

per cent. of all children with whooping-cough will have an associated case within the house within six months, and in about 13 per cent. the infection will presumably be conveyed by the first child to the second. It must be noted that these probabilities are based upon the whole population regardless of whether there are any other children at home liable to be attacked or not. Moreover they are founded on the assumption that reporting was complete, but though this was not the case and the totals are therefore too small,* the *ratios* of second to first cases would not, I think, be seriously affected.

From data given elsewhere [1] it appears that in Battersea the proportions of home contacts excluded from school at ages 5 and 6 who were attacked by whooping-cough after a few days but within six months were respectively 24/195, or 12·3 per cent., and 17/200, or 8·5 per cent., and if these are increased in the ratio 22 : 13 to include the risk of simultaneous infection, they become approximately 21 per cent. at age 5, and 14 per cent. at age 6. Half the mean annual attack rates recorded in children as a whole at ages 5 and 6 were respectively 3·51 and 1·75 per cent. The risk of contracting whooping-cough at ages 5 or 6 was therefore increased six- or eight-fold by the occurrence of a case in the same house. At pre-school ages the relative risk would no doubt be much greater, since the children who were not home contacts would not be exposed to the danger of school infection. The Holborn data showed [8] that of 721 children under 10 who were home contacts and who had not previously had whooping-cough, and who had also escaped any signs of attack during the first three days from onset of the first case, 196 were attacked within six months. Increasing this total in the ratio 22 : 13 to include simultaneous infections, it follows that about 45 per cent. of children under 10 *not previously attacked* would be expected to have whooping-cough concurrently with or within six months of onset of a case in the house, that is to say, about half would escape by natural or latent immunity.

### House-to-house Infection

The distribution of intervals between the onset of whooping-cough in children living in neighbouring houses provides a clue to the importance of house-to-house infection. In Table XI. the result of analysing the intervals between cases in houses in the same street in Greenwich whose numbers differed by 2, 4, 6, 8, is shown for comparison with the distribution of intervals in the same house. Since the numbering of the great majority of streets is alternate, and of a small minority consecutive, houses whose numbers differ by a small even integer would, except in a few instances of end houses in consecutively numbered streets, be on the same side of the street. Where the difference in number was 2, houses would in most instances be next door to each other, but in some instances next door but one; where the difference was 4, houses would be mostly next door but one; but in some instances next but three, and so on. By grouping together houses differing in number by 11, 12, 13 . . . 20, a control group of houses was obtained which would be either on the opposite side of the street, or at least six houses away on the same side.

TABLE XI

*Frequency of Intervals between Cases in Neighbouring and Distant Houses (Greenwich 1919–29)*

| Interval. | Same house. | Same street, with numbers differing by— | | | | | Houses in different streets paired at random. |
|---|---|---|---|---|---|---|---|
| | | 2. | 4. | 6. | 8. | 11–20. | |
| 0–3 days .. .. | 381 | 2 | 1 | 2 | 3 | 10 | 1 |
| 4–28 days .. | 395 | 18 | 8 | 8 | 9 | 59 | 7 |
| 4–13 weeks .. | 97 | 8 | 11 | 15 | 11 | 76 | 16 |
| 3–6 months .. | 38 | 8 | 7 | 10 | 8 | 56 | 17 |
| 6–9 „ .. | 10 | 12 | 8 | 5 | 4 | 41 | 12 |
| 9–12 „ .. | 6 | 5 | 10 | 8 | 4 | 42 | 8 |
| 1–2 years .. | 64 | 28 | 33 | 29 | 19 | 168 | 50 |
| 2–3 „ .. | 56 | 16 | 25 | 20 | 25 | 138 | 48 |
| 3–4 „ .. | 67 | 21 | 31 | 19 | 24 | 165 | 34 |
| 4–5 „ .. | 46 | 20 | 23 | 25 | 17 | 170 | 34 |
| 1–5 years .. | 233 | 85 | 112 | 93 | 85 | 641 | 166 |

The frequencies were then corrected to allow for the limited range of time over which the records were available, thus whilst practically every case during the decade was at risk to be followed by another, one day later, only about 8/10 of them were at risk to be followed by another, one year later, since 2/10 occurred during the last year of the records. The factors by which the actual frequencies had to be divided in order to correct for this are shown at the foot of Table XII. The corrected frequency distributions of intervals between cases when expressed in terms of the corrected frequency of intervals of 1–5 years, a number which could not be in any way affected by infection, will then indicate whether there was a greater risk of infection passing between children in neighbouring houses than between children in houses at a greater distance apart, and also how the risk changes with lapse of time. These are shown in Table XII.

Considering first the relative frequencies of 4–28 day intervals, which give the best index of the risk of house-to-house infection, for houses at a distance in the same street this was four times that expected if incidence of cases was at random; for houses whose numbering differed by 4, 6, or 8 it was also four times that expected, but for houses numbered two apart, most of which would be next door pairs, the risk was 9·8 times that expected. This shows that in Greenwich the risk to a child of contracting whooping-cough is more than twice as great if there is a case next door as it is if there is a case in some other house in the street, but is not appreciably greater if there is a case two or three doors away. When there is a case in the same house the risk is about seven times as

* I have shown [1] that in Battersea about two-thirds of the actual incidence at all ages is recorded.

TABLE XII

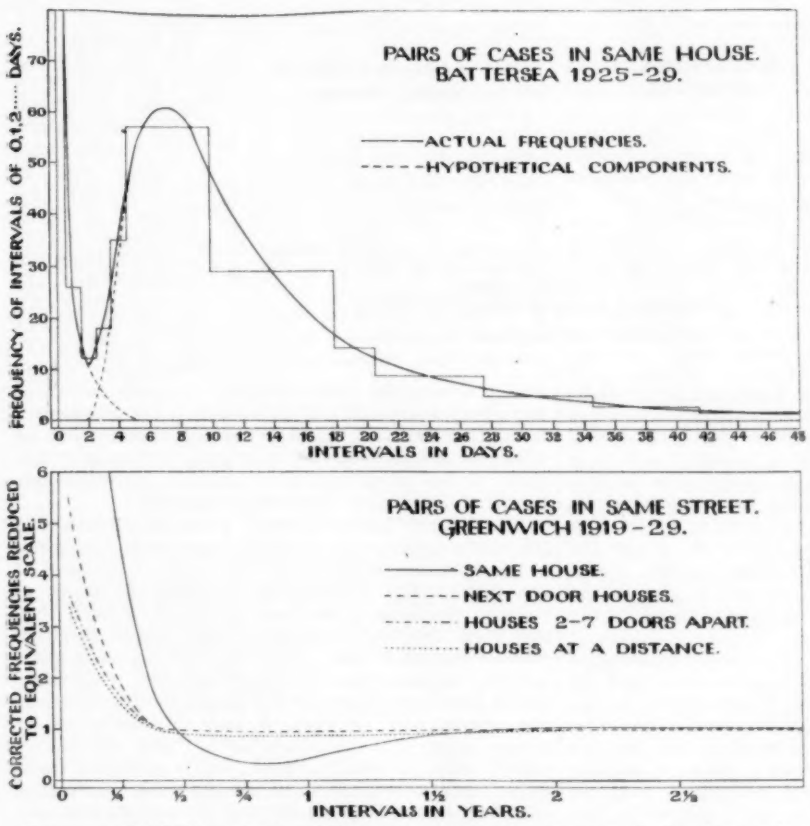
| Houses in same street. | Corrected frequency of intervals in terms of mean corrected frequency for 1–5 years, reduced to an equivalent range. | | | | | | |
|---|---|---|---|---|---|---|---|
| | 4–28 days. | 0–3 months. | 3–6 months. | 6–9 months. | 9–12 months. | 1–2 years. | 2–5 years. |
| Pairs in same house | 71·42 | 36·87 | 1·77 | 0·49 | 0·31 | 0·86 | 1·05 |
| Pairs in houses with numbers differing by 2 | 9·77 | 3·38 | 1·06 | 0·97 | 0·94 | 0·99 | 1·00 |
| Pairs in houses with numbers differing by 4, 6, or 8 | 3·93 | 2·53 | 1·03 | | | | |
| Pairs in houses with numbers differing by 11–20 | 4·19 | 2·40 | 1·02 | 0·80 | 0·85 | 0·89 | 1·03 |
| Houses in different streets, paired at random | 1·94 | 1·55 | 0·91 | | | 1·03 | 0·99 |
| Correction factors for duration of records | 1·000 | 0·974 | 0·884 | 0·833 | 0·802 | 0·761 | 0·614 |

great as when there is a case next door, or 18 times as great as when there is a case in some other house in the street.

At intervals longer than three months there is no evidence of any appreciably greater risk of infection from next door than from any other house in the street. There is still a risk of infection within the same house, however, as noted above. At 6–12 months' interval there is only half as great a risk that a second child will develop whooping-cough in the same house as that a child will develop it in some other house in the street, whilst at 1–2 years the risk is virtually the same. This forms part of the evidence, given elsewhere,[1] leading to the conclusion that a transient latent immunisation of contacts, protecting them for about a year, occurs. These results are more clearly depicted in the lower part of Fig. 1. As a further check a few houses in different streets were paired at random; with the results shown in Tables XI. and XII. The results do not differ significantly from those for houses at a distance in the same street except that at intervals of 4–28 days the risk is less, suggesting that infection is conveyed between children living in the same street even though they are not living next door.

In the lower portion of Fig. 2 the mean epidemic curve for the nine wards of Battersea in 1928–29, obtained by bringing together the months of maximal incidence in each ward, measuring the time scale forwards and backwards from this and totalling the numbers of cases in each corresponding month on the scale, is compared with the curve for the whole borough, for which the maximal month was December. The amount of dispersion on the time scale is not appreciably diminished by thus comparing the mean epidemic in a single district with that in the borough as a whole—that is to say, although the epidemic wave does not run quite concurrently in the neighbouring districts, it does not move about to the same extent as measles, but is more widely diffused over the whole borough at any given time.

FIG. 1




Frequency distributions of intervals between onset of successive cases of whooping-cough.

## Infection within Schools

In the upper part of Fig. 2 a similar procedure has been followed in the Wandsworth epidemic of 1926–27 in order to arrive at a mean epidemic curve for 21 of the largest schools, and when this curve is compared with that for the borough as a whole, after reducing each to the same total of 1000 cases, there is a notable contrast. The mean school curve rises and falls again much more rapidly than does the curve for the borough as a whole, and reaches a much higher peak. This must indicate that infection is more freely carried from one child to another, and the epidemic comes more quickly to an

end within a given school population than within the population as a whole.

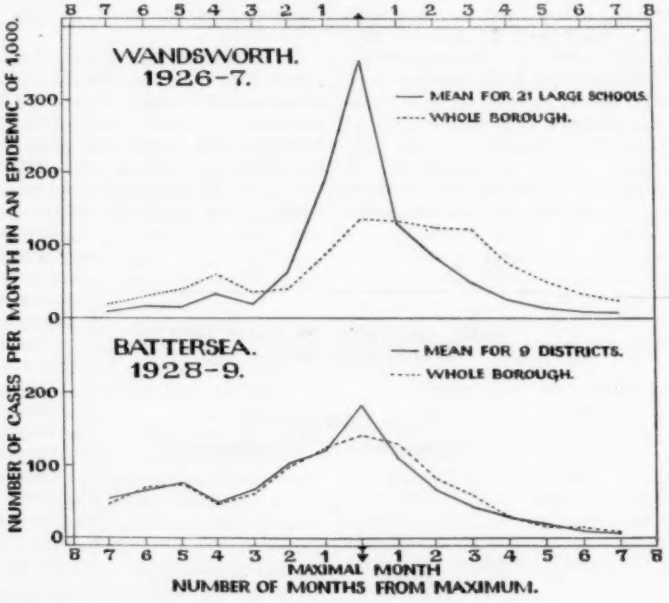
How is this accounted for? No preventive measures are used except to exclude home contacts. If these were kept at school the epidemic in the school would obviously come to an end just as quickly, perhaps more quickly, and it seems necessary to conclude that the epidemic ends by practically all the susceptible children at school being attacked or rendered immune for the time being. Outside the school, however, the epidemic goes on amongst the younger children almost unchecked, and because they are more scattered it takes a longer time to reach the same end.

### Possibility of Control

The question arises whether the practice of excluding from already infected schools home contacts to whooping-cough cases does any good, whether in fact it is not actually harmful. Most of these children spend their time playing with children of pre-school age, and since only an occasional supervision of these children is possible, and the most infectious stage precedes the onset of the whoop, many of them may infect younger children. If every susceptible child attending an infected school is going to either have an attack or develop a latent immunity in any case before the epidemic runs its course, and it is difficult to doubt that in face of the evidence, these children would be better at school until the first sign of catarrh or cough, when they could be immediately sent home and the mothers instructed to keep them indoors. Owing to the much greater danger of whooping-cough to young children it should be our aim, as in measles, to delay as long as possible the attack by seeking to protect the children of pre-school age from infection by school-children. In justification for this it is sufficient to notice that the fatality-rate in Battersea fell from 4 per 100 cases at age 0–1 to 0·2 per 100 at ages 5–10. Street infection has been shown to play some part in the spread of infection, and this might be reduced by the action suggested above.

Within the home it is also worth while to attempt protection, for although complete isolation may be impossible, a sufficient degree of separation during the most infectious stage may be practicable to so dilute the amount of infection to which the younger children are exposed as to enable them to acquire the necessary temporary latent immunity to secure them from the immediate danger, supposing the theory to be correct. The contention that "they had better get it over whilst they are young" is not a justifiable excuse for saving trouble to parents and school authorities, because in addition to the rapidly diminishing danger to life and to subsequent health with advancing age, there is a reasonable chance, of the order two in five, that if a child escapes immediate attack he or she may escape it altogether. Possibly the use of convalescent serum or parent's blood for home contacts may eventually provide, as in measles, a method of securing the occurrence of a mild attack which will produce a permanent immunity, a more satisfactory result than the mere production of temporary immunity, and in this case isolation within the house would become not only unnecessary but illogical.

FIG. 2




Whooping-cough epidemics in London.

### Conclusions

(1) The notification records of whooping-cough during the last decade in the metropolitan boroughs of Greenwich, Battersea, Wandsworth, and Holborn combined show an excess of female over male incidence amounting, after correction for changes in sex ratio with age in the populations at risk, to 5 per cent. at ages up to 4 years, increasing to 20 per cent. by the eighth year and 50 per cent. by the tenth year. The excess was considerably greater in Greenwich than Battersea. At ages under 7 there was in Greenwich no excess of girls amongst cases commencing from October to January, but an excess of about 20 per cent. amongst cases arising during the other months. The sex ratio was not significantly different in secondary cases in houses from that in first cases. The age-distribution of whooping-cough incidence is slightly different in the rising phases of epidemics, a somewhat larger proportion of children aged 3 to 6 being attacked.

(2) Children aged 4, 5, and 6 are more liable to infect other children in the same house with whooping-cough than are children of other ages. A study of the distribution of intervals between the onset of cases in the same house leads to the conclusion that the most frequent interval is one week, that the probability of a case occurring with a fortnight's interval is about half as great as this, with three weeks about one-fifth, with a month's interval about one-eighth, and between 3 and 6 months afterwards about one two-hundredth. The incubation period may, it would appear, be as short as three days, but is most likely to be about a week. Infection after six weeks is probably due to an exacerbation of the infective condition brought about by a super-

imposed catarrhal infection. About one case in every hundred is followed by another case in the same house after an interval between six weeks and six months.

(3) The risk of contracting whooping-cough at ages 5 or 6 was increased six- or eight-fold by the occurrence of a case in the same house, and at pre-school ages it would be increased to a greater extent. The risk, at all ages under 10 years, of contracting whooping-cough within a month was, in Greenwich, seven times as great if a case occurred in the same house as if one occurred next door. The risk of contracting whooping-cough within a month was more than twice as great if there was a case next door than if there was a case in some other house in the street, but was not appreciably greater if there was a case two or three doors away. About 45 per cent. of children under 10 who have not had whooping-cough may be expected to contract it concurrently with, or within six months of the onset of, a case in the same house.

(4) Infection between children living in the same street commonly occurs, and the risk of it is greatly enhanced for children living next door to one another. School epidemics tend to come to an end more quickly than in the surrounding district, and it seems probable that practically all the susceptible children at school are either attacked or rendered temporarily immune. The value of excluding from infected schools home contacts to whooping-cough cases is questionable; it would probably be better to keep these children at school until the first sign of catarrh or cough, and then send them home to be kept indoors. It is worth while, if at all practicable, to attempt isolation of a child with whooping-cough from other children within the home during the most infective period, particularly if any of these contacts are under 4 years of age, since this may increase the chance of the remaining children acquiring temporary latent immunity or escaping infection, and postponing the attack until an age when the disease is less dangerous.

(5) The proportions of children known to their parents to have had whooping-cough before entry to school at ages 3, 4, 5, and 6 are greater in those boroughs of London where the population is less dense and better housed. Though this may be partly accounted for by more accurate observation and diagnosis in such areas, it is evident that increased crowding of the population does not in different districts of London result in increased incidence of whooping-cough, but rather the reverse, a phenomenon difficult to account for unless latent immunisation occurs.

Towards the expenses of this research a grant was made by the British Medical Association. I am indebted to Miss Maud Kirby for drawing the diagrams.

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MANCHESTER CANCER HOSPITAL.—The Manchester and Salford Hospital Saturday and Convalescent Homes Fund has endowed a bed in the new Cancer Hospital at Manchester at a cost of £1000.

## MEDICINE AND THE LAW

### Royal Veterinary College: Libel Action

SOME five years ago Capt. Hobbs undertook to raise £20,000 for the Royal Veterinary College at Camden Town, which had fallen on evil days. This sum was thought sufficient to provide a special department for dogs, including a hospital for non-infectious canine patients, an operating theatre, and perhaps a special lecture theatre. Capt. Hobbs was particularly careful to obtain assurances that no part of the money would be used for any vivisectional purpose. Mainly by founding the Tail Waggers' Club and by organising National Dog Week he raised the required sum with the aid of thousands of contributions from dog-lovers. Strange as it may seem, his campaign met with bitter opposition. The College was licensed as a place where experimental work might be done; this fact caused the movement to be suspect. Miss Blanche Watson, propaganda secretary to the local branch of the British Union for the Abolition of Vivisection, wrote a vigorous letter to a Hull newspaper during the second National Dog Week in 1929. She warned dog-owners that by entering their pets as Tail Waggers they were encouraging vivisection. "The long and the short of the business," she wrote, "is to build and maintain the vivisection laboratories at the Royal Veterinary College. National Dog Week and its outcome, the Tail Waggers' Club, are simply masquerading under the cloak of humanitarianism, their real object not being the welfare of the dog but its exploitation." She quoted a statement from a London newspaper that the object of the Club was "the thrusting of torturous experimentation by the arch-exploiter man" upon hapless creatures whose consent was neither asked nor given. She mentioned no names, but Capt. Hobbs (sometimes described as the Tail Wagger-in-Chief) naturally complained that the accusation meant that he was a liar and a fraud. He brought an action against Miss Watson for libel, and he called witnesses who said they thought her letter referred to him and they gathered the impression that he must be a hypocrite. Under cross-examination he agreed that many people felt strongly (as he did himself) about the vivisection of dogs, that the College was registered as a place for vivisection experiments, that some of its professors held licences for that purpose, and that there was a research institute "just across the lawn from the College." This institute, he said, had been entirely excluded from his consideration when he was raising the fund. All these questions to him were directed by Miss Watson's counsel in order to establish her plea that her letter was fair comment on a matter of public interest. Sir Frederick Hobday, principal and dean of the College, explained that it had been arranged that no part of the fund should be used for what was known as vivisection. He described the work of the College, its educational side, and its busy "out-patients' department" to which poor people brought their animals. The research institute was under Government control, with a different governing body from that of the College; the money for the institute came wholly from the Ministry of Agriculture. The defendant in the witness-box declared her abhorrence of vivisection. In 1929 she saw it stated that the National Dog Week was held to endow a chair of research. She understood the statement to mean investigation into human and animal diseases by experimentation on the living animal. To this she was strongly opposed. When

she wrote her letter she knew nothing of Capt. Hobbs and had no intention of injuring him. In cross-examination she admitted that she thought the organisers of National Dog Week and the Tail Waggers' Club were deliberately misleading the public as to their intentions. She agreed that she meant to convey that they were getting money from the public by untruths and false pretences. She did not believe that any good had been done by human or animal inoculation. Asked as to the discovery (by means of inoculation) that rats were carriers of bubonic plague, she said it was a quite unnecessary exploitation of the rat because plague could be investigated by other means. In all research work, she said, there was torturous experimentation on living animals. Mr. Justice Branson told the jury that Capt. Hobbs, who was not named in the letter, had no right to complain unless it was understood to apply to him. If the jury thought the letter did imply that he was a hypocrite and had obtained money by the false pretence that he meant to benefit the dogs, whereas his intention was to submit them to vivisection experiments, then the jury might think the letter was defamatory. On the question of damages the judge said that, if the jury thought the plaintiff was entitled to succeed, they would consider the fact that Miss Watson rushed into print without taking the elementary precaution of asking Capt. Hobbs what the real object of the fund was. The jury found for the plaintiff and awarded £500 damages. Judgment was given for this sum with costs.

The limits of fair comment are generous. "Every latitude," once observed Lord Esher, "must be given to opinion and to prejudice." However wrong the opinion expressed may be in point of truth, or however prejudiced the writer, it may still be within the prescribed limits. But the defence of fair comment can be countered if the jury take the view which they took of Miss Watson's letter. She did not, it seems, attempt to justify what she had said. Every vivisection controversy invariably arouses strong feelings. At the beginning of the plaintiff's action, Capt. Hobbs's counsel announced that the trial would involve acute contention between a protagonist of the Tail Waggers' Club and a convinced antivivisectionist; it would therefore be desirable that the jury should not include either persons who held strong views against vivisection or persons whose dogs were members of the Tail Waggers' Club. Two members of the jury were found to belong to the latter class; they left the box and were replaced. The judge confessed that his dog was a Tail Wagger, and, later in the case, when a reference was made to societies formed to combat antivivisection propaganda, he felt obliged to mention that he was himself a subscriber to the Research Defence Society. Nobody objected, however, to his trying the case. Miss Watson's counsel was not concerned to prove that the antivivisectionists were right, but merely to show that the subject was one of public interest about which people might be expected to feel keenly and to express themselves vigorously.

### Hospitals' Responsibility for Nurses

A recent Scottish appeal (Reidford *v.* the Lord Provost, Magistrates, and Councillors of Aberdeen) furnishes a valuable statement of the legal responsibility of hospital managers in Scotland in respect of a patient's claim for damages for alleged neglect on the part of a medical practitioner or nurse on the hospital staff. It also shows that the Scots law and the English law reach much the same result though by different principles.

Mr. Reidford, a commercial traveller, was an inmate of the Woodend Hospital, Aberdeen, on two occasions in 1930. He claimed damages against the Aberdeen corporation (as the authority responsible for the hospital) for the alleged carelessness and neglectful treatment which he said he received from the nursing staff. The Aberdeen sheriff court dismissed his action on the grounds that his averments were irrelevant and lacking in specification. This, if a southern lawyer may venture a paraphrase, means that firstly, even if the alleged facts were proved, they would fail to furnish a proper legal basis for a claim, and secondly, the claim failed to supply sufficient particulars—e.g., as to the names of the nurses—to give the hospital authorities a fair chance to meet the allegations. The judgment of the Lord President on the appeal to the Court of Session, well reported in the *Scotsman* of Jan. 28th, indicates that Mr. Reidford complained (*a*) that during his first fortnight in the hospital the nurses omitted to supply him with certain alkali powders at the regular and appropriate intervals as directed by the doctors, and (*b*) that the nurses did not on their own responsibility administer the laxatives which his condition required. Other and later allegations of inadequate food and inattention in the wards were apparently too vague to be considered.

Dealing with the legal position the Lord President first allowed himself the mild criticism that a similar Scottish case (Lavelle's case) which had recently been decided in the House of Lords had been dealt with by their Lordships on the lines of English decisions instead of in accordance with Scottish law. This is a point on which the Scottish courts, administering their own system of law based on that of Rome, are properly sensitive. The Lord President went on to explain the Scots law applicable. There was a complete and infallible test of the liability of hospital managers in the Roman distinction between two kinds of hiring contracts, *locatio operis* and *locatio operarum*. It is a feature of the contract classified as *locatio operis* that the employer is answerable for all defects whether due to his own want of skill and carelessness or that of his servants and subordinates. A *locatio operarum* exists where someone of professional skill is called upon to do and advise what his skill dictates and not what the employer orders: "the person who makes with such a man or woman a *locatio operarum* is not liable for the negligence of the professional person within the department of that professional person's function and skill." It was therefore hopeless to try to make the hospital managers responsible for alleged neglect on the part of the hospital doctors or surgeons whom they permit to exercise their professional functions in the hospital. "The reason is that in the performance of these duties the medical man or the surgeon is making available to the patients his services; he is not acting as the servant of the managers at all; they can neither give him orders nor control him within the limits of the performance of his professional skilled duty." There might be an action against the individual doctor or surgeon; that was another matter. In the Aberdeen case it was clear that the nurses were not, and could not be, acting as the servants of the managers: "they were acting within the department which their professional skill entitled them to act in"; if they were acting expressly on the doctor's orders they were still acting within the area of professional skill and function. "It would be a very dangerous doctrine," concluded the Lord President, "to suggest that the managers of an infirmary were to be supposed to

have laid down any rules with regard to any item of the medical treatment of anybody who came into their sick-house. All they undertake is to provide an efficient, well-heated, clean, wholesome sick-house and furniture for the reception of patients, and no doubt to provide the necessary medicines and food." They do not undertake to treat or to cure or to operate upon anybody who is admitted to their institution.

If we compare this judgment (so important at a time when a progressive hospital policy is making treatment available for increased numbers of the public) with the English law, we find much the same result. All members of the staff of an English hospital are personally responsible for damages due to their own negligence. But, if there is a question of suing the authorities responsible for the management of the hospital, there is a simple question of agency and supervision. The hospital is liable for the negligence of persons who are its servants in the sense that their work is subject to the supervision of the committee of management. The hospital is not liable for negligence on the part of physicians or surgeons, paid or honorary, while they are exercising professional skill in respect of which the hospital management has neither the power nor the competence to interfere. The English legal position was made clear in 1905 in Evans *v.* Liverpool Corporation, on a complaint by a patient in the city's fever hospital. It was held that the corporation merely undertook that the patients admitted to its hospital should have competent medical advice and assistance; having appointed a competent and duly qualified medical practitioner the corporation was not liable for his negligence. Thus English law and Scottish law reach the same result, though it may be that the Scottish law gives greater protection to the hospitals in cases where complaint is made against nurses—e.g., in injuries from hot-water bottles. English law would presumably ask whether the alleged negligence arose in work which the nurse was doing as servant of the hospital. The question whether she was in fact acting under the supervision or orders of the medical practitioner in charge of the case has cropped up more often perhaps in patients' claims against the practitioner than in their claims against the hospital. Scottish law would apparently be more ready to say that in all nursing work the nurse's actions would be within the area of professional function and skill and therefore independent of control by the hospital managers. If the Scots law is in fact more favourable, there seems good reason for asking that the English law shall fall into line.

## IRELAND

(FROM OUR OWN CORRESPONDENT)

### MEDICAL MEN IN THE DÁIL

STUDYING the full list of elections to the Dáil one finds that it contains the names of eight medical men. They are Sir James Craig, Drs. J. J. O'Reilly, J. Lynch, T. F. O'Higgins, R. E. Davitt, F. C. Ward, P. J. O'Dowd, and J. Ryan. Two of these—Drs. Ryan and Ward—held office in the Government. Only one of the medical deputies is coming fresh to the Dáil—namely, Dr. R. E. Davitt. Dr. Davitt is one of the physicians to Jervis-street Hospital, Dublin. Two medical men who have been deputies for several years—Mr. T. Hennessy and Dr. Myles Keogh—have failed to secure election. Both will be missed, for both were prominent figures, and Mr. Hennessy was generally regarded as expressing medical opinion in public affairs. He has an intimate knowledge of medical matters in Ireland, and particularly of the conditions of the public medical services. In his championship of the rights and privileges of the medical profession he never permitted himself to forget that the interest of the public is supreme. He was popular with all parties in the Dáil, and both there and outside his absence will be regretted.

## AUSTRALASIA

(FROM OUR OWN CORRESPONDENT)

### RECONSTITUTION OF NEW SOUTH WALES MEDICAL BOARD

SOMETHING of a stir has been caused by the Minister for Health, Mr. R. W. B. Weaver, reconstituting the New South Wales Medical Board. Members of the previous Board, who had given many years of honourable service, were communicated with by letter from the Minister, who stated that in very many directions there was a need for an entire rearrangement of the existing conditions. Further, that he had given consideration to the functions of the New South Wales Medical Board and, while deeply appreciative of the work of this honorary body, he considered the time had now arrived for a general reconstruction of the Board. To permit of this he deemed it expedient that the appointments of all members of the existing Board be terminated, and suggested that the members should tender their resignations.

The members of the Board met and communicated with the Minister, stating that they desired to deal with the letter as a Board on Dec. 14th, when they would furnish him with a reply. However, on Dec. 10th a statement appeared in the daily press purporting to emanate from the Minister and indicating that "about seven members of the Medical Board would have to retire on the policy adopted by him, for the reason that they had reached or were over the age of 65 years."

In the morning press of Dec. 14th it was announced "that Mr. Weaver had issued a minute which had been signed by the Government declaring vacant all seats on the Board, and had nominated seven doctors to form the reconstituted Board." A number of letters appeared in the daily press criticising the Minister for his action in this matter, the criticism being levelled not at the personnel of the new Board, but at the manner of dismissal and lack of courtesy to members of the previous Board.

## PARIS

(FROM OUR OWN CORRESPONDENT)

### AN EXAMINATION SCANDAL

THE suicide of a woman resident medical officer of a Paris hospital has given a tragic turn to a series of incidents which have not been without their lighter side. She was involved in the scandal of the recent examination for resident hospital appointments, and after being summoned, with all the other interns concerned, to give evidence before a representative of the public authorities, she had a nervous breakdown. This examination began last October, between 600 and 700 candidates presenting themselves for about 80 vacancies. In order to eliminate the possibility of the examiners being tempted to favour their own

pupils or other candidates towards whom they might feel a kindly partiality, the examination was conducted so as to assure the anonymity of the candidates. Their numbered but unsigned papers were read aloud one after the other to the examiners by a staff of interns, to which the doctor in question belonged. By sandwiching these readers aloud in between the examiner and the examinee, it was hoped that the former would not only be saved from the exhausting task of deciphering much spidery MS., but would also find it easier to pass objective judgment. But unfortunately there seems to have been a widespread belief among the examinees that extra points could be gained if clues to their identity could be furnished in their papers. The whispers about cheating grew into a murmur, and finally into such a general uproar that official action had to be taken by the Assistance Publique. The official inquiry led to the startling discovery that out of 620 candidates, as many as 282, or 45 per cent. of the total, had sent in one or more papers marked in some distinctive fashion. It has therefore been decided to cancel the original examination. A new examination will be held in a month or two, and new examiners will set new questions to the same candidates. If most of the candidates who were successful in the first examination are so again, a cogent answer will be forthcoming to the old objection that such examinations, even when conducted honestly, put candidates at the mercy of too big an element of chance. But this is a very big "if."

# THE DOCTOR'S DAY

"Come, tell me how you live," I cried,
"And what it is you do!"

## XIII.—AN INDUSTRIAL DOCTOR

By an "industrial doctor" I mean a doctor who is employed full-time in a factory. In order to give a more intimate picture of the life, I will give a brief description of the factory in which I work and the size of the medical staff. It should be understood that industrial medicine in this country is developing slowly, and that many factories now have clinics, varying in size from a part-time nurse to a complete staff of full-time doctors and nurses.

In the factory where I am employed we make foodstuffs and employ about 5000 workers, of whom rather more than half are men and boys. The medical staff consists of two doctors and three trained nurses, one of whom spends all her time sick-visiting. My clinic reckons to do any work which comes to it, but we do no visiting outside the factory, except (1) to those employees between the ages of 14 and 16 who have no panel doctor, and who ask us to visit them when they are unfit for work; (2) the visiting done by the nurse, which is more in the nature of social than medical work.

The largest proportion of work coming to my department is very similar to that done in a casualty department of a hospital. Much of the work is surgical in type and minor in nature—cuts, bruises, sprains, septic conditions, boils—but there is no limit to the severity of the conditions seen. I keep an emergency bag, and am always ready to do any necessary emergency operation in the factory. Several times I have stood by while a machine was taken to pieces in order to release a worker who was trapped in it and badly injured. Such happenings are, however, emergencies, and we are not fitted to deal with these major injuries throughout their course; we have, therefore, our own ambulance, and we can have any case into hospital within five minutes. On the other hand, moderately severe injuries, such as fractures and lacerated tendons, are not uncommon.

### THE MORNING AND ITS VICISSITUDES

By the time I arrive, about 9 A.M., my clerk has a list of all the people who have asked to see us, and we proceed to send for them. We get them in small batches, and this has to be carefully organised, because we do not want to waste the employees' and the firm's time by keeping people waiting unnecessarily; on the other hand, we cannot afford to be kept waiting ourselves, because we may have 100 or 150 patients to see, and we always get through this list before dinner-time. By so doing we leave the afternoon free for redressings, accidents, those people who may be taken ill for medical reasons, and for all the other work which we do, and which I shall describe later. Some of the cases will be surgical, some medical, some new, and some we have seen on the previous day. We treat all medical cases which come to us, provided they are fit enough to stay at work, and we do our own dispensing. We make a small charge to recover the bare cost of the drugs used and, with the exception of such things as vaccines, all other treatment is given free.

So my morning is spent. Perhaps it sounds fairly straightforward and simple; sometimes it is, and sometimes it is the very reverse. The average doctor in practice works more or less to his own time, and is almost completely his own master; in industry, on the other hand, production is always the master, and is ruthless in calling the tune. The work may be done in a constant series of interruptions and inquiries—perhaps about subjects which have only a faint relation to medicine—and one needs much patience and tact. My department has four telephones, and there are times when all of them seem to ring all day long. It can be very exasperating to have to break off in the middle of an examination of a chest to answer a telephone inquiry as to the food value and the metabolism of some article, when the information required is only wanted for advertising purposes. Again, the finest organisation in the world can break down and become chaos simply because several accidents have happened about the same time and have to be treated at once, in spite of the fact that a batch of patients we have just sent for have to be kept waiting. Then the great god of production is held up, and many are the questions which have to be answered. Remember that British industry is fighting for its life, and everything depends on a high output.

The department is kept open during the dinner hour by sending the nurses off in relays; this is done because the workers go to dinner at different times, and because many of them have dinner in our own canteen. The afternoon is taken up by all that other work which has to be done. The jobs are many and varied, and I will describe them briefly.

### VOCATIONAL AND OTHER EXAMINATIONS

All applicants for employment are examined. This does not mean that only absolutely fit workers are taken, but rather that many with physical defects are accepted and their owners placed on the work which they can do. This vocational placing can only be done if the doctor has a very detailed knowledge of the various jobs on which the workers are to be put. A record card is made at the time of the examination, and every visit the employee subsequently makes to the department, with all the necessary information, is entered on that record. Obviously, this vocational selection would rapidly become useless unless every worker passed through the department's hands every time his work was changed. In practice this is done; although the workers do not always have to be re-examined, because it is sometimes possible to tell if they are suitable by reference to their records.

There are, too, many special examinations which have to be made. I keep a list of between 50 and 60 of our higher executives, for whom I do a complete overhaul every 6 or 12 months. The firm believes

in prevention, and as these men and women are most important to them, they believe it pays to recognise and treat illness in its earliest stages. This is the whole idea behind the employment of a medical staff; such attention is not given for philanthropic motives, and these purely preventive examinations are illustrations of modern business methods.

Other examinations we do are (1) monthly weighing, questioning, and, if necessary, examination of all cases of tuberculosis which have come back to us after treatment. These cases may be kept on a monthly list for many years, depending on their progress, and are subsequently transferred to a three-monthly list; later, possibly, they are taken off that too. To show the value of this list, I would mention the case of one of my patients who was in a sanatorium in 1913, and from then has been seen periodically, in spite of the fact that he has had no return of positive symptoms. Last year he developed a cough with a positive sputum, and is now back in a sanatorium. (2) After an interval of about six months we re-examine many of the children who have come to us at 14 years of age. Many of them we recognised as having some defects when they came originally, and some of them we find have developed further defects at the end of this period. A large proportion of these children have not reached puberty when they come to the firm, and I think there can be no doubt that their health can be made or marred in the first few years of their industrial life. At the end of six months most of them are well established in their new life, and will continue to do well without fuss, but there are others who need careful watching. (3) Examination of those workers who have some health hazard peculiar to the industry. We have some who are working in fairly high temperatures, where the relative humidity is high, and where there is a good deal of noise. In the case of adult women and young workers of both sexes I regard this as a hazard, and I examine periodically. (4) Some workers are sent to us because they are not doing well at their jobs, and we have to find whether there is any medical reason for the failure. Frequently there is, but it is only arrived at after much searching, and I often feel that a training in psychology would be a great help to the industrial doctor. The reason for any particular patient's failure to adapt to his ordinary life may be deep-lying and far removed from his work, but the result is always seen in his work in the long run, and then becomes vital to him. I am sure that many of us these last few years will have noticed more and more failures of adaptation, with their concomitant nervous symptoms. (5) Insurance examinations for those workers who have been injured and are receiving compensation.

### THE DOCTOR AS INDUSTRIAL EXPERT

In addition to these examinations there is work to be done in the factory itself. I am sometimes consulted about the lay-out of new plant, and I am asked what particular type of worker will be needed for a new job—e.g., men, youths, adult women, boys, or girls—and what are the special qualifications necessary. I keep an eye on the ventilation, heating, and lighting of the rooms, and not infrequently I carry out tests, either to satisfy myself or because I have had a complaint about a room. It is essential that one must know how to use a kata-thermometer and a photometer. In addition, I am asked multitudinous questions by the management about every conceivable subject which has, or is thought to have, some medical aspect.

In our quieter moments we try to do some detailed investigations into subjects which are causing us some anxiety—such as sickness rates and the causes of lost time, with particular reference to dysmenorrhœa and functional nervous conditions. But the time available is usually all too short or is interrupted before the study has begun. In industry one has to be prepared to do anything at a moment's notice, and to do it quickly. We once did over 500 vaccinations in two days, in addition to our ordinary work, because there had been a case of small-pox in the factory. We felt that many cases of the disease in a food factory would mean ruin to the firm, and we vaccinated every possible contact. Happily we had no more cases.

During the last ten years I have been with the firm we have had 364,515 patients through my department, giving an average of 36,451 annually. These patients came entirely of their own accord, and it is only in exceptional cases—e.g., infectious or contagious diseases—that any pressure is brought to bear on them.

### THE CHOICE OF THIS CAREER

It might be asked whether I would advise industrial medicine as a career. I should have to give a qualified answer. The hours are good; I work from 9 A.M. until about 5.45 P.M. or 6 P.M., and I have no night work to do and I am free on Saturdays and Sundays. I suppose the hours are about the same as those worked in the public health service, but undoubtedly the security is not so great. In industry there seems to be little security for medicine; the science is a new one to them and is not admitted by all industrialists to be a paying proposition. There is therefore no guarantee that any particular firm will continue to employ a doctor, nor is there any guarantee that a firm will continue to be prosperous enough to employ sufficient people to make the services of a full-time doctor necessary. The work itself is very interesting, but to do it well I think a man should be interested in something more than medicine as a science; the number of cases we see of real scientific interest is not great, but one has to deal with many very interesting things, and I personally have acquired some knowledge at least of many interesting people. Industry itself, with its modern methods of efficiency, is an absorbing subject, and is never without its thrills and changes. In a word, I would not advise a doctor to go into industry permanently unless he is prepared to take a risk; for a young man it would not be without interest for a few years, and would certainly help him to get an intimate knowledge of his fellow-men.

## THE SERVICES

### ROYAL NAVAL MEDICAL SERVICE

Surg. Comdr. J. F. Ainley is placed on the retd. list at his own request.

### ROYAL NAVAL VOLUNTEER RESERVE

Surg. Sub-Lts. E. G. Brewis, P. M. Coats, and G. J. Murray to be Surg. Lts.

Proby. Surg. Sub-Lt. S. I. Ballard to be Surg. Sub-Lt.

### ROYAL ARMY MEDICAL CORPS

Lt.-Col. A. T. Frost retires on retd. pay.

Maj. A. B. H. Bridges is placed on retd. pay.

Maj. W. H. S. Burney to be Lt.-Col.

### TERRITORIAL ARMY

Capt. T. C. McKenzie to be Maj.

Lt. E. M. R. Frazer to be Capt.

G. M. Goodwille (late Cadet, Edinburgh Univ. Contgt., Sen. Div., O.T.C.) to be Lt.

A. MacDonald (late Cadet, George Watson's Coll. Contgt., Jun. Div., O.T.C.) to be Lt.

### INDIAN MEDICAL SERVICE

Capts. D. G. Karandikar and T. P. Rao relinquish their temp. commns.

### ROYAL AIR FORCE

Flying Officer D. C. MacGilchrist is promoted to the rank of Flight Lt.

The undermentioned are granted permanent commissions in the ranks stated: Flight Lt. C. A. Rumball and Flying Officers O. M. Fraser and A. M. Weston.

*Dental Branch.*—The undermentioned Flying Officers are promoted to the rank of Flight Lt.: J. G. Stewart and J. J. Lawson.

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HARLOW WOOD ORTHOPÆDIC HOSPITAL.—Although this Nottinghamshire hospital was opened only three years ago an enlargement, which will cost between £12,000 and £13,000, is already necessary. Last week the first sods were cut by the Duchess of Portland, the president, a colliery owner, and a miner who had been unable to walk for ten years but was completely cured in the hospital.

# PANEL AND CONTRACT PRACTICE

## Certification in Pregnancy and Post-confinement

THERE is considerable misunderstanding on the part of both insurance practitioners and approved societies as to the conditions under which benefit is properly payable during pregnancy and after confinement. The Ministry of Health in Memo. 324/A.I.C. point out that pregnancy is not sufficient in itself to entitle an insured woman to any cash benefit under the National Health Insurance Act. Sickness or disablement benefit can only be paid in respect of a period during which a pregnant woman is incapable of work. A normal, uncomplicated pregnancy would not ordinarily involve incapacity for work, though during its latest stages or in exceptional circumstances a woman might become incapable. A doctor is therefore debarred from giving a medical certificate in order to enable benefit to be claimed unless he is satisfied, as a result of an examination of the woman, that she is incapable of work. After the puerperium (of four weeks) a woman is not entitled to sickness or disablement benefit unless her physical or mental condition is such that she can properly be certified as incapable of work within the meaning of the National Health Insurance Act.

These statements sum up the position clearly, but it is easy to see how difficulties occur. Mrs. A walks into her doctor's surgery, asks for a certificate to go on the panel, stating that she is pregnant. Probably she adds one of the following reasons for her request: "My employers won't have me at work"; "I can't do my work any longer"; "My agent told me to get a certificate from you"; "My friend Mrs. B went on the panel when she was pregnant and I do not see why I should not go on too"; or "Dr. X gave my friend a certificate without any difficulty, and if you don't give me a certificate I shall change my doctor—the agent said I was to get a certificate." This last threat, of course, involves possible loss of confinement fees as well as panel fees, and probably private attendance on the non-insured part of the family—quite a serious consideration to many doctors. How is he to deal with the situation? The first thing to do is to examine the patient and to ascertain (1) the stage of pregnancy; (2) the kind of work; (3) the presence or absence of any physical defects, such as bad varicose veins, piles, œdema of legs, albumin in urine, excessive vomiting. If one of these disabilities are found a certificate can properly be given. If the pregnancy appears to be quite normal, but in its late stages, the kind of work will need special consideration. If before becoming pregnant she was a waitress, the question arises whether she can now act as a waitress without doing herself harm. If the doctor thinks she can, he should refuse a certificate even if she says her employers will not have her back. An exception to this rule is in Class K cases, where a woman marries and leaves off work but continues in benefit for a further year or more; here it becomes necessary to judge her capacity for work when pregnant from the standard of housework. Can she perform without harm ordinary daily household duties? Probably it is only in the very late stages of normal pregnancy that a woman will be unfit to do such work at her own rate, in her own way, though she might be unable to work away from home where she could not rest during the day.

The reason often given for demanding a certificate, that the employer will not have the pregnant woman back at work, is irrelevant to the payment of sickness benefit. If a woman is not incapable of work and her work is not unsuitable to her state, and yet the employer will not have her back, it is advisable for the doctor to write her a private note on a sheet of paper setting out the position. The approved society will then have the onus of deciding, as they do in cases like scabies or impetigo on the face, whether to pay benefit or not. Such cases are not legally incapable of work. On the other hand, the woman who says "I cannot do my work any longer," often has either some additional incapacity or some occupation which involves much stooping or bending, and these patients require careful examination.

To doctors the most annoying statement a patient can make is that she was told by the agent of her society she was to get a certificate. This has led to much acrimonious talk between doctors and societies, but it is quite easy to see what happens. The agent of the society cannot say yes or no to a question whether a woman is entitled to sick pay. What he does say, often under instruction from his head office, is, "You must get a certificate from your doctor." This phrase is interpreted by the patient to mean that the doctor is bound to give a certificate, and this is what she tells the doctor. Thus a situation which causes much irritation and administrative difficulty all round is really due to a mistake in the interpretation of words not easy to avoid among uneducated people. Sometimes doctors use expressions which patients interpret incorrectly, such as "I will give you a certificate so that you can get your money," or "Take this certificate to your secretary, he will pay you then." The patient assumes that the doctor knows what is due to her, though the agent or secretary may be under orders from head office to stop payment for some administrative infringement or other reason unknown to the doctor.

It is not unknown, however, for agents to go round advising women to claim when pregnant, and then advertising the successful claims in order to encourage recruitment of new members. Fortunately this is not a common proceeding, and most of the bigger societies have given definite instructions to their agents in order to remove any erroneous impression. The real cause of difficulty in competitive areas is a threat of change of doctor. A tired doctor may protest that he can ill afford to risk losing private cases, confinement fees, as well as panel fees, in order to carry out the letter of an Act of Parliament run by a Minister who is always worrying him about records and prescriptions in order to save the funds of some approved society whose officers rarely have a good word to say for his work. It is easy for him to believe that a patient feels much worse than her physical signs show, and to convince himself that it would be wrong to refuse to put her on the panel. In time of course a doctor will realise that a weak attitude leads to panel slavery, and that patients will not retain much respect for a doctor who does not hold to his own judgment in these matters. These cases can usually be handled tactfully so that the doctor gets his own way and the patient is nevertheless satisfied. If the incapacity really is in doubt the case can always be referred to the regional medical officer for a report.

The certificate of post-confinement cases is not so difficult. For four weeks after her confinement she gets maternity benefit and is not eligible for sick pay or disablement benefit. After this period she is not entitled to such benefits unless she is certifiable as incapable of work. In this case a first certificate should be given and not an intermediate one. The commonest causes of such incapacity are backache and anæmia.

The certification of pregnancy cases has given rise to much discussion in approved society circles as well as in medical committees. It has been suggested that no sickness pay should be given during pregnancy, but that a larger maternity benefit should be paid in its place. This would be rather hard on the woman who broke her arm when pregnant, and required funds to help finances to carry on. Again, it has been suggested that sickness pay should only be given for those ailments which are not connected with pregnancy, an almost impossible position for any doctor to determine. The matter is certainly very important. It has recently been brought to the notice of the panel committee for London by the organising assistant secretary in his quarterly report, and not for the first time.

### Surcharging

In THE LANCET of Jan. 14th we expressed agreement with the Glasgow critics that "it is a pity there have been no cases of investigation of under-prescribing." By this we intended to convey that there had been no cases tried by panel committees where the charge was under-prescribing as distinct from over-prescribing. But we are informed, and are glad to point out, that regional medical officers do interview practitioners whose average is definitely below the general average. It is usual to find such cases either in country areas where the doctor is a dominating personality, who has little faith in the efficacy of drugs, or in areas in London, like Soho, where a large foreign population does not readily take medicine. Another type of practice where the average is low is that attached or connected with some industrial firm who habitually send their cases to hospital rather than to the panel doctor. It is obviously difficult to prove that a doctor is not giving sufficient medicines, even though his prescriptions may be well below the real average, and that is probably why no cases have yet been taken as far as a panel committee for investigation.

# CORRESPONDENCE

## MASKED HYPERTHYROIDISM

*To the Editor of* THE LANCET

SIR,—Dr. J. R. H. Towers is to be congratulated on his paper on Masked Hyperthyroidism and Heart Disease in your issue of Jan. 14th. Those of us who are especially interested in thyroid problems are frequently confronted with the sort of case Dr. Towers describes in which certain phenomena such as tachycardia, wasting, nervousness, and tremor are present, but the thyroid gland is not visibly enlarged.

Dr. Towers, I am sure, does not need to be reminded that many goitres not visible are nevertheless palpable owing to their abnormal consistence, or to the fact that in a muscular neck a thyroid gland of two to three ounces in weight may not be visible, yet if the muscles are relaxed it may be felt. It is also true that the thyroid gland may be enlarged, yet neither inspection nor palpation, even by those skilled in this branch of medicine, reveal the fact. The normal thyroid gland weighs in the young adult, when it is at its maximum size, about four-fifths of an ounce, but a gland twice or even thrice this weight may be neither visibly nor palpably enlarged. Where Dr. Towers might, I think, enlighten us further is by giving details of the weights of thyroid tissue removed in those cases submitted to operation, and also by informing us as to the histological structure of the extirpated tissue. This would help to solve the problem whether thyrotoxicosis ever occurs with a thyroid gland which is normal in size or normal in structure, or both. I am convinced that the contrary proposition is true—viz., that thyrotoxicosis never occurs in association with a thyroid gland which is normal in size, still less in one which is normal in structure.—I am, Sir, yours faithfully,

Harley-street, W., Jan. 24th, 1933. CECIL A. JOLL.

## THE MIGRATION OF "MALTA FEVER"

*To the Editor of* THE LANCET

SIR,—In the discussion on Prof. W. E. Hume's paper on Undulant Fever at the Royal Society of Medicine on Jan. 24th, I submitted that undulant fever was new to Malta when first heard of in the fleet in 1856, and Dr. E. W. Goodall asked where it had been before that. Perhaps you will let me answer now—in South Russia. Surgeon Captain S. F. Dudley tells us it was then new to the fleet, and there is no trace of it among the diseases of Malta recorded by that careful physician, John Hennen, in medical charge there from 1821–25. An expedition was made to Kertch, at the foot of the Sea of Azof, during the Crimean War, and troops returning thence to Malta, on board men-of-war, seem to have infected the ships, and perhaps the island. It is doubtful if any reports of this disease were made from South Russia before the Great War, but several have appeared since, from Baku,[1] from Batoum [2] and from Rostoff, at the upper end of the Sea of Azof in 1931.[3] Brucella septicæmia is clinically so indefinite in men that it is not likely to be diagnosed unless attention is specially directed to it. It was recognised at Rostoff in 1925, but epidemic abortion of cattle it was caused particular concern. The first human *abortus* case was at last recognised in a veterinary surgeon, for three years concerned with epidemic abortion (loc. cit., ref. 3, p. 237).

*Br. abortus* and *melitensis* fevers—they seem scarcely distinguishable—are widely spread. We heard on the 24th that they are notifiable in France and Germany, cases are always being reported in new places; Prof. Hume told us of 17 unexpectedly found in the North of England, and Surg.-Lieut. Commander S. G. Rainsford [4] says that 20 per cent. of normal serums in Ireland agglutinate *Br. abortus*. So this septicæmia is almost as widespread, if not so virulent, as appendicitis, of which hardly anyone had heard 50 years ago. And other diseases have been known to migrate—yellow fever from West Africa to the West Indies, and in 1848 to South America (Bahia); malaria to Mauritius from India. Cholera, plague, and influenza migrate quite commonly, syphilis recently invaded Uganda, sleeping-sickness and chigoe were brought from West to East

[1] THE LANCET, 1924, i., 963.
[2] Ibid., 1930, ii.,, 671.
[3] Arch. f. Schiffs- u. Tropen-Hyg., 1931, xxxv., 81, 237.
[4] Brit. Med. Jour., Jan. 28th, p. 147.

Africa by Stanley's porters, and German authors hold that Indian immigrants brought tubercle to East Africa; so there are many parallel cases to the bringing of undulant fever from South Russia to Malta along the lines of human communication.

It is very difficult to trace modern diseases through the clinical descriptions of earlier writers. There is a special difficulty with undulant fever, for which our chief "token," if I may use that word, is rheumatism. I can well remember the severity of the rheumatism that affected some of the cases of "Malta fever" I saw between 1886–89, but not for years after that did I hear of gonorrhœal rheumatism. Did the latter exist in those earlier days? And how is one to discriminate between them in the old annual reports before they are officially discriminated?

I am, Sir, yours faithfully,

W. E. HOME,
Fleet-Surgeon.

Southborough, Jan. 29th, 1933.

## "DIAGNOSTIC EXPLORAL SUCTION" OF THE NASAL SINUSES

*To the Editor of* THE LANCET

SIR,—May I plead for the usage of "exploral suction" (*syn.* suction exploration) or some equally descriptive term in recording the diagnosis of sinusitis based on samples of sinus contents withdrawn by suction into a syringe for macroscopic and bacteriological investigation?

The diagnostic technique of exploral suction is now widely used in this country and in America, yet often enough the rather vague term "exploration" is employed without qualification or any indications of the special method adopted. It is true that I myself long continued to use the term "exploration" (connoting "searching out") which I applied originally, when towards the close of the last century I introduced this method to replace that of washing out discharge in the sinus through the nasal passages—e.g., from the maxillary antrum by Lichtwitze trocar, now commonly referred to as proof puncture. However, the term exploration is now being used by other laryngologists in another sense—e.g., for proof puncture washing out—and hence, particularly in the diagnosis of sphenoidal or posterior ethmoidal sinusitis, it becomes desirable to employ a term conveniently short, and which implies that the samples on which the diagnosis is based have been sucked out separately from each sinus. It seems to me a pity that because a vague term has hitherto been used that there should be no clearer indication, in records, that particular pains have been observed to suck out the samples for investigation, instead of running them into the nasal passages and collecting for examination whatever issues from the nostrils, and which is bound to be contaminated.

Certainly, I should welcome suggestions of some better word for common usage than exploral suction or suction exploration, but as "exploration" has been very widely used in rhinology for 40 years the nearest variant to answer the purpose has advantages.

I am, Sir, yours faithfully,

PATRICK WATSON-WILLIAMS.

Clifton, Bristol, Jan. 27th, 1933.

## THE USE OF SYNTHETIC CREAM IN KETOGENIC DIET

*To the Editor of* THE LANCET

SIR,—In the ketogenic diet treatment of pyelitis it is essential to produce an intense ketosis during the first few days. If such a ketosis is not obtained, further treatment is often of little value.

The diet usually recommended contains a daily ration of 70 g. of protein, 250 g. of fat, and 20 g. of carbohydrate, and while it frequently succeeds with patients who can take exercise it sometimes fails with those confined to bed. More consistent results are obtained in these latter cases if the daily carbohydrate is reduced to 10 g. The problem is how to offer such a diet in a form acceptable to a patient who may be seriously ill.

Owing to the restricted protein intake, a large proportion of the fat must be taken as cream, in soups, fruit fools, ice-cream, cream jellies, and coffee. At Queen Charlotte's we allow 14 oz. of 40 per cent. cream per day. This amount of cream ordinarily contains 11 g. of lactose. If, however, a synthetic cream prepared from butter and 3 per cent. casein solution, with the aid of a cream mixer, is used, all the lactose, except for the trace present in butter, is eliminated. There is a clear saving of 10·5 g. of carbohydrate, and it is then a simple matter to supply a daily diet with 70 g. of protein, 250 g. of fat, and only 10 g. of carbohydrate, in a reasonably palatable form. The urine of a patient on such a diet contains ketones on the first day, and in many cases as much as 0·2 per cent. of aceto-acetic acid by the third or fourth day. If the 20 g. allowance of carbohydrate is adhered to, the sugar saved in milk and cream can be supplied more usefully in the form of extra fruit and vegetables.

The British Drug Houses, Ltd., supply a Light White Casein which is easily soluble and gives a neutral solution.—I am, Sir, yours faithfully,

A. T. FULLER.

Bernhard Baron Memorial Research Laboratories,
Queen Charlotte's Maternity Hospital, W.,
Jan. 26th, 1933.

## HUMAN TUBERCULOSIS OF BOVINE ORIGIN

*To the Editor of* THE LANCET

SIR,—Dr. Esther Carling's letter in your last issue inspires me to place before you some aspects of the problem which have not received the attention they deserve.

The continued mortality in man from tuberculosis of bovine origin is a lasting disgrace to the medical profession, and yet we continue to discuss it rather academically, and with the same detachment that we might give to the incidence of leprosy in the Middle Ages. The source of infection—milk—is clearly defined and generally acknowledged, yet 2000 persons, mainly children, die annually from this source of disease, and ten times that number are crippled or made ill for the same reason. We are all working to eliminate tuberculosis from cattle, which would, indeed, banish bovine tuberculosis from humans; but with a 40 per cent. incidence of the disease among cattle this laudable process may be expected to take 40 years, during which time something short of 80,000 persons, mainly children, will have died, and 800,000 be crippled or ill.

Those that regard the problem as more urgent advocate universal pasteurisation, pending the advent of detuberculisation, but this needs supervision, it is expensive, it is probably inapplicable to rural areas where the disease is most prevalent, and to educate the public to the right point and secure adequate pasteurisation would take at least ten years, during which time something short of 20,000 persons, mainly children, will have died and 200,000 be crippled or ill.

Yet the solution of the problem is really simple—milk should be boiled in each individual home before consumption.

Ten years ago when Dr. Evrot was sent to this

country by the French Comité National de Défense contre la Tuberculose to tell British tuberculosis workers what was being done in France, he was asked how they were meeting the problem of bovine tuberculosis, and he replied: "There is no bovine tuberculosis among the French people. Every household boils the milk." Recently I have been informed that the same custom prevails in Italy, and a long-standing member of the Indian Medical Service has also told me that he encountered no bovine tuberculosis among Indians because they boiled their milk—and we could do the same. Later, when we have eliminated disease from the cattle, boiling may be abandoned, but to-day there is urgent need for its adoption. Pasteurisation is expensive and uncertain, boiling is individual and certain.

It may be urged that such a step diminishes the food value of milk, as no doubt it also diminishes the value of other foods which it is the custom to cook. But there is no definite evidence of disease among children due to boiled milk, and moreover pasteurisation has much the same effect. It may also be said that mothers would not boil the milk, but I cannot believe that there would be more difficulty than in France or Italy. We have only to make up our minds to advocate the measure whole-heartedly and it would be done. Six months' organisation, six months' propaganda, and bovine tuberculosis would cease to occur in man.—I am, Sir, yours faithfully,

Paignton, Jan. 30th, 1933. ERNEST WARD.

## THE PRESCRIPTION OF THYROID

*To the Editor of* THE LANCET

SIR,—With reference to the article in your issue of Jan. 28th on the prescription of thyroid, may I say, on behalf of my members, who are all dispensing chemists, that the new B.P. by clarifying the position in regard to thyroid has rendered a valuable service. I was therefore glad to see your article, as it is necessary that the attention of the medical profession should be directed to the change.

Prescriptions for thyroid have been a source of considerable annoyance to dispensing chemists in the past because of doubts as to the doctor's intentions, and sometimes the doctor must have been disappointed with the results obtained with thyroid, simply because he has ordered "gland dosage" when he meant "dried gland dosage."

A general observance of the new style in regard to the ordering of thyroid will be a real boon to dispensing chemists.—I am, Sir, yours faithfully,

G. A. MALLINSON,
Secretary, National Pharmaceutical Union.
4, Queen-square, W.C., Jan. 30th, 1933.

*To the Editor of* THE LANCET

SIR,—Your annotation on the prescribing of thyroid gland preparations calls attention to a real source of danger. In view of the fact that the dried gland is many times stronger in thyroid substance than the fresh gland, there has often been a possibility for doubt on the part of the dispenser as to which the practitioner had in mind. Both prescriber and dispenser, therefore, should welcome the clear definition of the dried substance in the new British Pharmacopœia, which will go a long way to clear up doubts and to save a reference back to the prescriber.

The description in the British Pharmacopœia includes all the usual names employed in prescribing thyroid. It is to be hoped that medical men will note that a prescription written under any of these names will be interpreted in the future to imply that thyroid of the British Pharmacopœia is intended. My firm are prepared to accept this definition, and our dispensing establishments are equipped with supplies on the new basis. It will, of course, take a little time for doctors to accustom themselves to the change. Both kinds of preparation will therefore for the time being be kept in stock on our dispensing counters. The standardised Pharmacopœia preparation would appear to be the obvious basis for prescribing thyroid, and in our judgment the change indicated should be beneficial all round.

I am, Sir, yours faithfully,

E. T. NEATHERCOAT,
Managing Director, John Bell and Croyden, and Savory and Moore, Ltd.
Welbeck-street, W., Jan. 31st, 1933.

## THE MINIATURE HOSPITAL

*To the Editor of* THE LANCET

SIR,—It is to be hoped that the model of a hospital which is on view in New Bond-street, and which was described at some length in your issue of 28th inst., will not be regarded as the best British architects can achieve in hospital design. To the layman the general appearance is doubtless attractive, but there are various shortcomings which are at once apparent to anyone experienced in hospital management.

Examine, for instance, the ward for adult patients and its annexes. There is neither medicine cupboard nor cupboard for lotions, bowls, &c., both of which should have been recessed into the wall. No provision has been made for an instrument steriliser. An electric light should have been fixed over each bed for attending to patients when the curtains are drawn at night. There is no housemaid's cupboard for the storage of brooms and other cleaning materials, which will inevitably be hidden behind the bathroom or lavatory door. There is no fire-escape staircase. The linen cupboard is small, apparently unheated, and has antiquated shelving. There is no suitable ventilated accommodation for the receptacles for soiled and foul linen, unless the solitary sanitary dustbin in the corner of the ward is intended for this purpose!

And so one could go on, but enough has been said to emphasise the necessity for the closest collaboration between architect, matron, and doctor when planning a new hospital.—I am, Sir, yours faithfully,

West Middlesex Hospital, Isleworth, Middlesex, Jan. 31st, 1933. J. BASIL COOK.

## THE LATE DR. G. C. PARKIN

*To the Editor of* THE LANCET

SIR,—Mrs. Parkin, the widow of the late Dr. Parkin, of Malton, Yorkshire, gave birth to a daughter on Jan. 31st.

The result to date of our appeal, which is still open, for the education of Dr. Parkin's two children has been the sum of £770 13*s*. We wish to thank all those who have so generously contributed, and express our appreciation of the wide response we were able to obtain through your kindness in publishing our letter. It has been decided to hand over the final sum collected to the Royal Medical Benevolent Fund, the Committee of which has very kindly consented to act as trustees of the money. Contributions may still be sent to Mr. James Crooks, F.R.C.S., at the Hospital for Sick Children, Great Ormond-street, London, W.C.1.—We are, Sir, yours faithfully,

H. A. T. FAIRBANK, F.R.C.S.,
JOSEPH W. LEECH, F.R.C.S.,
W. T. GORDON PUGH, M.D.

Jan. 31st, 1933.

# OBITUARY

## SIR WILLIAM TAYLOR, K.B.E., C.B., F.R.C.S.I.

REGIUS PROFESSOR OF SURGERY, UNIVERSITY OF DUBLIN

WE regret to announce the death of Sir William Taylor, Regius professor of surgery in the University of Dublin, which occurred suddenly on Sunday last, Jan. 29th, in the sixty-second year of his age. He had shown no signs of illness, and the news of his death came as a shock to his friends.

Sir William Taylor was born in Donegal and was educated at the Strabane Academy, the medical schools of the Irish Royal Colleges, and the University of Dublin. He qualified with the double diploma of the colleges in 1893, having proved himself a distinguished student by securing the gold medal in operative surgery and the Mayne scholarship. Later, at the University of Dublin, he graduated in Arts and took the M.B. degree in 1901, having previously secured the F.R.C.S. Irel. In 1898 he was appointed assistant surgeon to the Meath Hospital, where he had formerly been interne, and two years later full surgeon to the institution. He was consulting surgeon to this hospital at the time of his death, so that he had given it life-long service.

SIR WILLIAM TAYLOR

[*Photograph by Elliott and Fry*

He soon established a well-earned reputation as an operating and consulting surgeon, and some of his experiences formed the subject of valuable communications to the *Transactions* of the Royal Academy of Medicine in Ireland and to the *British Medical Journal*. Elected to the Council of the Royal College of Surgeons in Ireland in 1914, he became vice-president, and two years later president, his term of office coinciding with the middle period of the war. On the outbreak of the war he was appointed, with the rank of colonel A.M.S., consulting surgeon to the Forces in Ireland, a position which he held for six years. He was one of the surgeons to the Dublin Castle Red Cross Hospital, and in 1917, at the request of the War Office, he organised a hospital in France, selecting the staff from men too old for ordinary service. The staff consisted almost entirely of members of the visiting staffs of the Dublin hospitals, every hospital being represented, and the "83 (Dublin) General Hospital" at Boulogne did admirable work at the closing period of the war. Taylor himself spent three months at the hospital, and his energy and skill were recognised with the C.B. in 1917 and the K.B.E. in the following year. Since that time his deep concern for the condition of ex-Service men has been expressed in many directions, and he was a notable figure among the group of Irishmen who went to Belgium and France to unveil memorials to their dead countrymen.

In 1923 Sir William Taylor became surgeon to Sir Patrick Dun's Hospital, in succession to Prof. Edward Henry Taylor, whom he also succeeded in the Regius professorship, the chair which he was occupying at the time of his death. He recently visited Canada and the United States and was received there warmly, the University of McGill making him an honorary LL.D., and the American College of Surgeons an honorary fellow. He was president of the Association of Surgeons of Great Britain and Ireland in 1924-25, and was elected president of the Royal Academy of Medicine in Ireland in 1927. In addition to his posts at the Meath Hospital and Sir Patrick Dun's Hospital, he was consulting surgeon to the Coombe Lying-in Hospital, the Steevens Hospital, and the Dental Hospital of Ireland. He was a D.L. for the county of Dublin, and held high rank in the Masonic Order. Few men can have combined so many important posts or acquitted themselves in their multifarious obligations with greater ability.

Sir William Taylor married, in 1898, Katharine, daughter of the late Dr. Hamilton Walker, and is survived by one daughter and three sons.

Our Dublin correspondent writes: "William Taylor was not only a leading surgeon in Dublin, but he was one of the outstanding personalities in the profession. Clear-headed, forcible, direct, he had made his way by his natural gifts of intelligence and character. As a surgeon he was careful in his diagnosis and confident in his judgment; in operation cool, quick, resourceful. Such a thing as 'nerves' was unknown to him. It was characteristic of him that he chose to perform major operations on his own family rather than refer them to other surgeons. As a teacher he was clear, precise, and entertaining. In public professional affairs he took a leading part, and his qualities of common-sense, quickness of understanding, and directness were of great value in shortening many a discussion and in expediting business. He had strong likes and dislikes, and he took no trouble to conceal either."

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## WILLIAM BLACK JONES, M.D. Lond.

William Black Jones, whose death is announced in Cardiff on Jan. 27th, at the age of 68, was well known in connexion with medical hydrology. He received his medical education at St. Bartholomew's Hospital, and qualified with the English double diploma in 1892. He graduated in the same year as M.B., B.S. in the University of London with honours in obstetrics and surgery, and acted as house surgeon and assistant in the throat department at St. Bartholomew's. He proceeded to the M.D. degree in 1894, and shortly afterwards began a long connexion with the Spa at Llangammarch. The waters of this spa contain barium to a somewhat unusual extent, and the fact was brought to the attention of the medical profession by Lauder Brunton, who suggested that the therapeutical properties of barium might be employed with advantage in certain cardiac disorders. Accordingly a considerable study of the matter was made at Llangammarch by various experts, but the spa, however, now relies for its reputation on the value of its mild saline waters in digestive catarrhs. While at Llangammarch Black Jones made some interesting meteorologic studies; one upon the measurement of sunshine at our health resorts attracted considerable attention, bringing home to the medical profession in England that they were behind

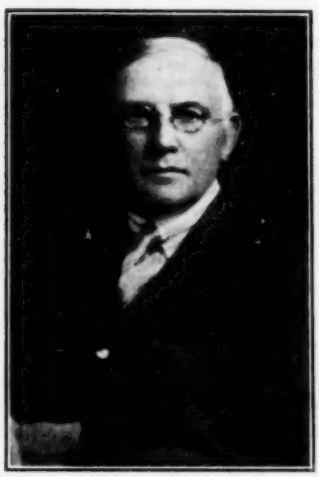
their continental brethren in demonstrating the amenities of their own health resorts.

Dr. Black Jones had retired from practice when he died. In addition to his post at Llangammarch, he was at one time M.O.H. for the combined Builth Wells and Colwyn urban and rural districts, and J.P. for Brecon. He was consequently a well-known man, and his death is widely regretted.

## CHRISTOPHER MARTIN, M.B. Edin., F.R.C.S. Eng.

WE regret to announce the death of Mr. Christopher Martin, the well-known gynæcologist, which occurred at his residence at Edgbaston, Birmingham, on Jan. 28th, after a long period of indifferent health.

Christopher Martin was born at Stockton-on-Tees in 1866 and educated at the Middlesbrough High School, whence he proceeded to the University of Edinburgh. Here he graduated M.B., C.M. in 1887, with first-class honours. He became resident surgeon at the Royal Maternity Hospital, and was for a time demonstrator of anatomy at the Edinburgh School of Medicine. He continued his studies at St. Bartholomew's Hospital and took the F.R.C.S. Eng. in 1891, having already formed his valuable professional connexion with that forceful curious character, Lawson Tait.



MR. CHRISTOPHER MARTIN

[Photograph by Beaufort

Christopher Martin had been introduced to Lawson Tait by Sir A. R. Simpson, whose house surgeon he had been in Edinburgh. Tait, some 20 years previously, with the vigorous coöperation of Mr. Arthur Chamberlain, and in the face of opposition from the Birmingham General and Queen's Hospitals, had founded in Birmingham a special hospital for women. To this hospital, now the Midland and Birmingham Hospital for Women, Martin was appointed assistant surgeon, and shortly afterwards entered into partnership with Tait, of whose professional and social life he later wrote an interesting record which was recently reviewed in our columns (THE LANCET, 1932, i., 1264). Tait's was a meteoric career, and his surgical prowess, which brought him so swiftly to the top, failed to sustain him there. Shortly after Martin joined him Tait resigned his many important appointments and retired into private life. But by this time Martin had secured by his own good work a high place in his special department of medicine. He was appointed assistant to the professor of gynæcology in Queen's College, Birmingham, and gynæcologist to the Birmingham General Dispensary, and the experiences of his large practice, institutional and private, were put out in practical papers on abdominal surgery and gynæcology, some of which were published in our columns, notably one on the treatment of myomata. He was one of the first in England to perform a total hysterectomy, and was successful also in extensive plastic operations for advanced cases of carcinoma involving the whole vagina. In 1902 he delivered the Ingleby lectures at the University of Birmingham upon the retention of the menses, and shortly afterwards became president of the Midland Obstetric and Gynæcological Society. Increasing calls upon him made no difference to his abiding interest in the Hospital for Women, with the progress of which he had been bound up since his first appointment to the institution more than 40 years ago, and it is in this connexion in particular that he has established a claim to permanent recollection in the city. He was one of the first fellows of the British College of Obstetricians and Gynæcologists.

Christopher Martin was married in 1920 to Miss Mary Clarke, M.B., Ch.B. Birm., who had been anæsthetist to the Hospital for Women, and held other medical appointments in Birmingham hospitals. He is survived by his widow.

Mr. Frederick Edge, a personal friend of 40 years' standing, writes: "On Christopher Martin's arrival at Birmingham with his mother I became acquainted with her to whom he always admitted how much he owed. He lived with her when he was a student in Edinburgh, while she possessed a wide knowledge of medical affairs, acquired as matron of the Middlesbrough Hospital, a post which she obtained when she was left a widow. The devotion of mother and son was striking. . . . On the many journeys which I made abroad, to France and Belgium, with Martin and other medical friends, he was a great guide, always producing excellent maps and notes on all points of historic interest, so that we came back carrying bags bursting with articles collected by the way. Martin could never do enough to please and entertain the party, and he always brought the tools necessary to remove the appendix of anyone who should require his assistance. The suffering of the incurable always aroused Martin's particular pity, and he was associated with the late Prof. Taylor in instituting the Taylor Memorial Home for Incurables, of which institution he was chairman until the end of his life. He always thoroughly enjoyed the renewal of old friendships, and with Dr. Douglas Stanley originated in Birmingham an Edinburgh Graduates' Dinner. He was chairman of the Gertrude Myer Home for Convalescents, and he enjoyed the organisation of picnic excursions for nurses and students. His was a truly generous nature."

## INFECTIOUS DISEASE

### IN ENGLAND AND WALES DURING THE WEEK ENDED JAN. 21ST, 1933

*Notifications.*—The following cases of infectious disease were notified during the week:—Small-pox, 16 (last week 18); scarlet fever, 1950 (last week 1739); diphtheria, 821; enteric fever, 12; acute pneumonia (primary or influenzal), 3781 (last three weeks 2017, 2816, and 3456); puerperal fever, 27; puerperal pyrexia, 139; cerebro-spinal fever, 61 (last two weeks 38 and 47); acute poliomyelitis, 2; acute polio-encephalitis, 1; encephalitis lethargica, 10; dysentery, 6; and ophthalmia neonatorum, 75. No case of cholera, plague, or typhus fever was notified during the week.

The number of cases in the Infectious Hospitals of the London County Council on Jan. 24th–25th was as follows:—Small-pox, 40 under treatment, 4 under observation (last week 48 and 3 respectively); scarlet fever, 1676; diphtheria, 1642; enteric fever, 9; measles, 270; whooping-

cough, 575; puerperal fever, 22 (plus 6 babies); encephalitis lethargica, 228; poliomyelitis, 3; "other diseases," 318. At St. Margaret's Hospital there were 13 babies (plus 10 mothers) with ophthalmia neonatorum.

*Deaths.*—In 118 great towns, including London, there were no deaths from small-pox, 3 (0) from enteric fever, 37 (0) from measles, 13 (1) from scarlet fever, 47 (12) from whooping-cough, 24 (6) from diphtheria, 42 (7) from diarrhœa and enteritis under 2 years, and 1589 (338) from influenza. The figures in parentheses are those for London itself.

The towns which reported the largest number of deaths from influenza were as follows (last week's figures being given in parentheses): Manchester 106 (49), Bristol 59 (24), Stoke-on-Trent 53 (15), Liverpool 52 (41), Leeds 49 (33), Sheffield 49 (14), Birmingham 38 (91), Bolton 29 (15), Nottingham 26 (9), Portsmouth 25 (35), Newcastle-on-Tyne 25 (36), Middlesbrough 21 (8), St. Helens 19 (6), Salford 18 (6), Coventry 18 (16), Bradford 16 (6), Leicester 16 (7), Walsall 16 (7), Stockport 15 (0), Sunderland 15 (13), Barnsley 15 (13), Gateshead 14 (19), Hull 13 (2), Doncaster 13 (10). In Greater London the areas chiefly affected were West Ham 28 (14), Ealing 15 (3), Tottenham 13 (5), Walthamstow 11 (4), Croydon 11 (6), and Leyton 10 (7).

Twelve of the fatal cases of measles were in Birmingham, and 3 each in Leicester, Cardiff, and Hull. Whooping-cough caused 3 deaths each in Hendon, West Ham, Liverpool, and Manchester. Four of the deaths from diarrhœa and enteritis under two years were in Liverpool, and 4 in Coventry.

The number of stillbirths notified during the week was 294 (corresponding to a rate of 47 per 1000 total births), including 46 in London.

# PUBLIC HEALTH SERVICES

## Influenza

IN the week ending last Saturday (Jan. 28th) the number of deaths from influenza in the 118 great towns of England and Wales was 1934, compared with 303, 681, 1041, and 1589 in the previous four weeks. In London the number was 370, compared with 25, 93, 242, and 338. In Scotland the decline in mortality continued, and the 16 principal towns reported 84 deaths, compared with 88, 111, and 108 in the three other weeks since the beginning of the year. In Glasgow, Edinburgh, and Aberdeen, where the deaths numbered 11, 17, and 5 respectively, the infection is steadily abating, and this applies to most of the big Scottish towns. The main exception is Dundee, where during the past three weeks there has been an equally steady increase and 26 deaths were reported last week.

The third week in January, for which fuller particulars are now available, witnessed a further extension of the outbreak, geographical as well as numerical. Of the 118 great towns of England and Wales only 2 failed to report any deaths from influenza, and the total increased, as already stated, to 1589. The general trend of mortality is shown by the fact that while 83 towns reported an increase, only 27 showed a decrease; in 8 the number of deaths was stationary. Some of the principal increases are noted in a neighbouring column in our weekly review of Infectious Disease. The geographical distribution of mortality is seen in Table I. (from the Registrar-General's Weekly Return), which gives the death-rates from influenza (per 1000 per annum) for the several weeks in the large towns of the country. It appears that in the week ended Jan. 21st there was a rise in every area except the eastern. The highest rate was in the northern area, not the midlands as before, and Greater London was again placed fourth, with a rate below the average. The eastern and south-western areas, and Wales, proved to be still relatively fortunate. As regards the age-distribution of the deaths, Table II. shows a slight but steady increase of mortality in the 45–65 age-group, while deaths at 0–25 have fallen to less than 10 per cent. of the total.

TABLE I

| — | 118 great towns. | South-eastern area. | Greater London. | Northern area. | Midland area. | Eastern area. | South-western area. | Wales and Monmouthshire. |
|---|---|---|---|---|---|---|---|---|
| 1932 Dec. 17th | 0·22 | 0·17 | 0·17 | 0·26 | 0·27 | 0·21 | — | 0·15 |
| ,, 24th | 0·30 | 0·13 | 0·17 | 0·35 | 0·69 | 0·10 | 0·23 | 0·23 |
| ,, 31st | 0·77 | 0·39 | 0·31 | 0·76 | 1·95 | 0·10 | 0·23 | 0·38 |
| 1933 Jan. 7th | 1·73 | 1·22 | 1·04 | 1·70 | 3·65 | 0·42 | 0·57 | 0·38 |
| ,, 14th | 2·64 | 2·56 | 2·36 | 2·78 | 3·25 | 1·78 | 0·91 | 0·98 |
| ,, 21st | 4·04 | 3·73 | 3·63 | 4·81 | 4·32 | 1·36 | 1·81 | 1·58 |

TABLE II

| — | Ages 0–25 | | 25–45 | | 45–65 | | 65+ | | Total deaths |
|---|---|---|---|---|---|---|---|---|---|
| | Deaths. | Per cent. | Deaths. | Per cent. | Deaths. | Per cent. | Deaths. | Per cent. | |
| 1932 Dec. 17th | 15 | 17·6 | 9 | 10·6 | 21 | 24·7 | 40 | 47·1 | 85 |
| ,, 24th | 23 | 19·2 | 19 | 15·8 | 34 | 28·3 | 44 | 36·7 | 120 |
| ,, 31st | 43 | 14·2 | 70 | 23·1 | 89 | 29·4 | 101 | 33·3 | 303 |
| 1933 Jan. 7th | 75 | 11·0 | 143 | 21·0 | 214 | 31·4 | 249 | 36·6 | 681 |
| ,, 14th | 112 | 10·8 | 196 | 18·8 | 336 | 32·3 | 397 | 38·1 | 1041 |
| ,, 21st | 156 | 9·8 | 311 | 19·6 | 525 | 33·0 | 597 | 37·6 | 1589 |

Influenza has spread rapidly southwards through France and Belgium, and has now reached Italy and Greece. Germany was affected comparatively late, and the *Deutsche medizinische Wochenschrift* of Jan. 27th (p. 143) said that although the notifications of respiratory disease had been rising there was as yet little statistical evidence of an outbreak in Berlin. The number of cases notified to the Allgemeine Ortskrankenkassen (general district sick-clubs) of Berlin from Jan. 23rd–30th show a subsequent rise, being 85, 150, 219, 215, 202, 253, and 393 daily. A statement by the health section of the League of Nations, issued through a press agency at Geneva, says (according to a summary in the *Daily Telegraph* of Jan. 27th) that the epidemic originated in Kansas, U.S.A., in the middle of December, and spread through the eastern states. Thence it reached Scotland before descending upon England and crossing to the continent.

FIRE AT A HOSPITAL.—On Sunday night, Jan. 22nd, a fire broke out in a lift at Birkenhead Infirmary as the result of the fusing of a wire in the electric generator. Since the hospital contained some hundreds ofpatients the entire strength of the Birkenhead fire brigade was requisitioned, and the flames, which had been attacked by the hon. medical and other staffs, were speedily subdued.

# MEDICAL NEWS

## University of Cambridge

On Jan. 27th the following degrees were conferred:—

*M.D.*—C. J. Scott,* I. C. C. Tchaperoff, and J. C. Ainsworth-Davis.
*M.B. and B.Chir.*—A. B. Carter and P. T. Ashby.
*M.B.*—M. H. Masina.
*B.Chir.*—G. A. Mandow,* F. H. Morrell,* W. D. Dick,* R. D. Roper, F. W. Shepherd, Eric Bradbury, A.R.R. Le Fleming, E. B. Pawson, R. H. Foster, and G. D. Wedd.

* By proxy.

## Royal College of Physicians of London

At a quarterly meeting of the College on Jan. 26th, with Lord Dawson, the president, in the chair, Dr. Stanley Barnes was elected a councillor in the place of the late Dr. Carey Coombs.

The following candidates, having satisfied the censors' board, were admitted to the membership:—

Arthur John Priestley Alexander, M.D. Belf.; Hyman Philip Brody, L.R.C.P.; Frank Storey Cliff, M.D. Lond.; Ronald Wilson Fairbrother, M.D. Manch.; Clive Hamilton Fitts, M.D. Melb.; John Macpherson Harker, M.B. Lond.; Howell William Howell, M.D. Lond.; Robert Andrew Hunter, M.D. Manitoba; Evan Jones, M.B. Lond., L.R.C.P.; Prince Mohan Kaul, M.B. Punjab; George Durant Kersley, M.D. Camb., L.R.C.P.; Parshotam Lal Khosla, M.B. Punjab; Ranald Ian Macdonald, M.D. Dalhousie; Noel Francis Maclagan, M.B. Lond.; Donald Uvedale Owen, M.D. Liverp.; Arthur Ewart Parks, M.D. Toronto; William Phillips, M.B. Wales, L.R.C.P.; Wilfrid Michael Priest, M.B. Lond.; Cedric Barrington Prowse, M.B. Camb.; Thomas Henry Pullar, M.B. Sheffield; William Hutton Scobie, M.B. Glasg.; Ronald Bodley Scott, M.B. Oxf.; Frederick Harwood Stevenson, M.D. Lond.; Thomas Scott Stone, M.B. Lond.; Edward Gwynne Thomas, M.B. Lond.; Hubert Carey Trowell, M.D. Lond.; John Francis Varley, M.B. Camb.: and Joseph Viccars Walker, M.B. Birm.

Licences to practise were conferred upon 134 candidates (118 men and 16 women) who have passed the final examination of the Conjoint Board and have complied with the by-laws. The following are the names and medical schools of the successful candidates:—

G. Abbey, Leeds; P. H. Addison, St. Mary's; Monica H. Austin, King's Coll.; Margaret M. Baddeley, Liverp. and West London; T. A. Baldwin, Univ. Coll.; R. Barnes, M.B., Manch.; A. M. F. Batty and A. A. W. Beach, L.D.S., Birmingham; G. F. Benjamin, Liverpool; R. Bennett and J. G. Bentley, St. Bart.'s; A. Birnbaum, London; R. W. Blakey, Leeds; Joan C. D. Bolt, Royal Free; R. W. W. Bose, Camb. and London; F. C. Bourgault du Coudray, London; A. Bowen-Davies, Camb. and Guy's; W. D. Bower, Camb. and St. Thomas's; D. J. T. Brinkworth, Middlesex; J. Britanischski, M.D. Berlin, London; H. W. Burge, King's Coll.; R. J. Buxton, Camb. and St. George's; Jean W. D. Calman, Univ. Coll.; B. S. Carter, St. Thomas's; T. Chadderton, Manchester; H. F. Chard and H. K. Childs, St. Thomas's; M. M. Cohen, M.B., Manch.; D. D. Coleman, Coll. Med. Evang. and Charing Cross; D. M. Cooper, St. Thomas's; Esther Copperman and H. Creditor, Univ. Coll.; F. J. Curtis, Camb. and Guy's; J. E. Davenport, Camb. and London; J. Davies, Cardiff and London; D. J. Dawson, St. Bart.'s; H. De, Calcutta and St. George's; Ursula D'Monte, M.B. Bombay, West London; E. J. Dennison, St. Thomas's; L. S. De Silva, King's Coll.; M. E. Disney, London; A. G. Donald, Guy's; J. F. H. Eagles, Bristol; G. J. Evans, Guy's; J. C. G. Evans, St. Thomas's; L. H. Evelyn and E. D. Falconer, Guy's; S. T. Falla, London; Catherine F. Forrest, Royal Free; Getel Gimpelson, King's Coll.; I. H. Gosset, Oxford and St. Thomas's; E. N. Hailey, Birmingham; P. P. Halemani, Bombay and Middlesex; J. L. Hamilton-Paterson, Univ. Coll.; B. A. E. Harley, Middlesex; Freda M. B. Harmer, Royal Free; E. H. C. Harper, St. Thomas's; H. R. Hartnell and B. T. W. Harvey, London; P. G. F. Harvey, St. Bart.'s; Anne E. M. Herbert, King's Coll.; A. Hilmy, Cairo and St. Bart.'s; F. G. Holman, Guy's; J. M. Holmes, M.B., Leeds; R. A. Hooper and W. R. S. Hutchinson, Camb. and St. Thomas's; L. G. Irvine, Camb. and London; L. S. Jaikaran, London; A. G. Johnson, Guy's; E. G. Jones, Cardiff and London; Grace M. Jones and Nellie E. Jones, Cardiff; S. O. Jones and G. E. Kerr, King's Coll.; H. B. D. Kettlewell, Camb. and St. Bart.'s; F. B. Khambatta, Bombay and West London; F. R. Kilpatrick, Cape and Guy's; B. H. Kirman, King's Coll.; S. B. Lavine, Jefferson and Middlesex; J. A. E. Lawn, St. Bart.'s; M. Lee, St. Thomas's; Beatrice Lewis, Univ. Coll.; M. B. Lindsay, St. Thomas's; A. E. Locke, Guy's; Madeleine Lorsignol, Royal Free; J. F. Lown, Camb. and St. Bart.'s; B. McArdle, Guy's; J. O. McDonagh, Univ. Coll.; B. M. McOwan, St. Bart.'s; G. A. Mais, St. Thomas's; S. O. Massey, Middlesex; S. F. Mitchell, Univ. Coll.; G. R. Morgan, R. J. G. Morrison, and L. R. Norsworthy, St. Bart.'s; Josie M. Oldfield, Royal Free; C. V. Oldroyd, Manchester; J. P. O'Shea, Guy's; E. D. Page, St. Mary's; E. B. Pawson, Camb. and St. Bart.'s; K. M. A. Perry, Camb. and London; A. G. Potts, Guy's; L. E. J. Poulier, Ceylon and St. George's; G. A. Ransome, St. Bart.'s; P. B. Rao, Madras and London; F. W. Roberts, Middlesex; H. J. M. Robinson, Camb. and St. George's; A. H. W. Roffey, King's Coll.; R. D. Roper, Camb. and St. Bart.'s; R. H. Rushton, King's Coll.; A. G. Salaman, Camb. and London; A. W. Sampey, London; S. S. Selladurai, Ceylon and Middlesex; Arthur Gray, Middlesex; F. W. Shepherd, B.Chir. Camb., St. Bart.'s; B. W. Smith, Guy's; G. S. Steele-Perkins, Camb. and Guy's; K. W. Stroude, M.B., Liverp.; G. H. Stuart, Univ. Coll.; T. V. Tattersall, Camb. and Manchester; W. E. Thomas, Cardiff; R. E. S. Turner, Oxford and St. Bart.'s; H. G. Ungley, M.B., Durh.; E. J. Walsham, St. Thomas's; S. P. Wanchoo, Punjab and Middlesex; G. L. Ward and C. J. T. Watson, Camb. and St. George's; E. E. S. Wheatley, St. Thomas's; W. H. Whiles, King's Coll.; I. Whittington, St. George's; J. Wilton, Witwatersrand and St. Mary's; V. F. Forbes Winslow, St. Bart.'s; E. J. S. Woolley, St. George's; and Freida Yarmalinsky, Univ. Coll.

The Milroy lectures, on Pneumococcal Infections, will be delivered by Dr. Robert Cruickshank at the College on Feb. 23rd, 28th, and March 2nd; the Goulstonian lectures, on the Physiology of the Gall-bladder and its Functional Abnormalities, by Dr. Charles E. Newman, on March 7th, 9th, and 14th; and the Lumleian lectures, on Complications of the Specific Fevers, by Dr. Charles R. Box, on March 16th, 21st, and 23rd.

## University of London

At a meeting of the senate held on Jan. 25th Mr. L. N. G. Filon, F.R.S., Goldsmid professor of applied mathematics and mechanics in the university, was elected vice-chancellor for the remainder of the year 1932–1933, in the place of the late Mr. J. L. S. Hatton.

The degree of D.Sc. (in human anatomy) was conferred on Dr. Francis Davies, university reader in anatomy at King's College, and (in physiology) on Dr. E. W. Fish, assistant dental surgeon to the Royal Dental Hospital and St. Mary's Hospital. The title of reader in experimental physiology in the university was conferred on Dr. H. P. Gilding, senior lecturer in physiology at University College.

Among those appointed Fellows of King's College was Mr. Eardley Holland, obstetric and gynæcological surgeon to the London Hospital. Mr. Holland was a student of the College from 1898 to 1904, and subsequently tutor in obstetrics and assistant obstetric surgeon to King's College Hospital.

Six lectures on Chemical Structure and Pharmacological Action—a study of drugs with specific actions on nerve-endings—will be given in the physiology theatre of University College, Gower-street, W.C., by Mr. H. R. Ing, D.Phil., at 5 P.M. on Mondays from Feb. 13th.

## Scottish Conjoint Board

As a result of recent examinations of the conjoint examining board of the Royal Colleges of Physicians and Surgeons of Edinburgh and the Royal Faculty of Physicians and Surgeons of Glasgow the following candidates have been granted the diplomas of L.R.C.P. Edin., L.R.C.S. Edin., L.R.F.P. and S. Glasg.:—

L. R. L. Solomon, Louis Danzis, M. O. Luck, George Kwryan, R. H. Payne, M. G. Myers, N. C. Smiedt, I. S. Lipschutz, E. L. Mommen, G. L. G. Major, A. A. Khan, C. A. S. O'Doherty, Jacomina A. Richter, J. G. Harrison, Aminuddin Ahmed, D. H. McNamara, Robert Keay, W. J. Silva, K. K. Retnam, Janet R. Hardy, C. W. Cook, Stewart McCallum, Noah Philips, A. L. Kahn, and J. C. T. Reid.

## Dr. Alfred Cox Testimonial Fund

The secretary to the committee organising this Fund, Dr. Rowland Fothergill, has issued the final balance-sheet giving the details of the artist's fee, the cost of the appeal, and the cost of the presentations. Reproductions of the portrait will not be undertaken, and subscribers have had returned to them the amount forwarded in respect of copies, but any subscriber who has not received back his remittance for a copy should communicate with Dr. Fothergill, 16, Brunswick-place, Hove, Sussex.

## British College of Obstetricians and Gynæcologists

A quarterly meeting of the council was held on Jan. 23rd, with Dr. J. S. Fairbairn, the president, in the chair. Dr. Henry Russell Andrews was elected a vice-president of the college, and Dr. Arthur E. Giles was elected honorary librarian. It was decided that the annual general meeting of the college should be held on April 5th in Birmingham, for the convenience of those attending the Congress of British Obstetrics and Gynæcology.

The following were formally admitted to the Fellowship: Cedric Lane-Roberts, Louisa Martindale, James Henry Drew Smythe; and the following to the Membership: Eric Arthur Gerrard, George Frederick Gibberd, Edith Hall, Alan Morris Johns, Richard Glyn Maliphant, Ellen Douglas Morton, William Joseph Rawlings, William Foster Rawson.

The following, having fulfilled the requirements of the by-laws, were elected to the Membership and will be admitted at the annual general meeting: William Saxon Barton, Alexander Broido, Jajneswar Chakravarti, Richard Victor Dowse, Wallace Freeborn, John Sydney Green, David McKay Hart, William Charles Wallace Nixon, James Vincent O'Sullivan, Robert Watson, Ralph Kuper White, Nellie Wilkes.

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# PUBLIC HEALTH SERVICES

## Influenza

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TABLE II

| — | Ages 0–25 | | 25–45 | | 45–65 | | 65+ | | Total deaths |
|---|---|---|---|---|---|---|---|---|---|
| | Deaths. | Per cent. | Deaths. | Per cent. | Deaths. | Per cent. | Deaths. | Per cent. | |
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| ,, 21st | 156 | 9·8 | 311 | 19·6 | 525 | 33·0 | 597 | 37·6 | 1589 |

Influenza has spread rapidly southwards through France and Belgium, and has now reached Italy and Greece. Germany was affected comparatively late, and the *Deutsche medizinische Wochenschrift* of Jan. 27th (p. 143) said that although the notifications of respiratory disease had been rising there was as yet little statistical evidence of an outbreak in Berlin. The number of cases notified to the Allgemeine Ortskrankenkassen (general district sick-clubs) of Berlin from Jan. 23rd–30th show a subsequent rise, being 85, 150, 219, 215, 202, 253, and 393 daily. A statement by the health section of the League of Nations, issued through a press agency at Geneva, says (according to a summary in the *Daily Telegraph* of Jan. 27th) that the epidemic originated in Kansas, U.S.A., in the middle of December, and spread through the eastern states. Thence it reached Scotland before descending upon England and crossing to the continent.

FIRE AT A HOSPITAL.—On Sunday night, Jan. 22nd, a fire broke out in a lift at Birkenhead Infirmary as the result of the fusing of a wire in the electric generator. Since the hospital contained some hundreds ofpatients the entire strength of the Birkenhead fire brigade was requisitioned, and the flames, which had been attacked by the hon. medical and other staffs, were speedily subdued.

# MEDICAL NEWS

## University of Cambridge

On Jan. 27th the following degrees were conferred:—

*M.D.*—C. J. Scott,* I. C. C. Tchaperoff, and J. C. Ainsworth-Davis.

*M.B. and B.Chir.*—A. B. Carter and P. T. Ashby.

*M.B.*—M. H. Masina.

*B.Chir.*—G. A. Mandow,* F. H. Morrell,* W. D. Dick,* R. D. Roper, F. W. Shepherd, Eric Bradbury, A.R. R. Le Fleming, E. B. Pawson, R. H. Foster, and G. D. Wedd.

* By proxy.

## Royal College of Physicians of London

At a quarterly meeting of the College on Jan. 26th, with Lord Dawson, the president, in the chair, Dr. Stanley Barnes was elected a councillor in the place of the late Dr. Carey Coombs.

The following candidates, having satisfied the censors' board, were admitted to the membership:—

Arthur John Priestley Alexander, M.D. Belf.; Hyman Philip Brody, L.R.C.P.; Frank Storey Cliff, M.D. Lond.; Ronald Wilson Fairbrother, M.D. Manch.; Clive Hamilton Fitts, M.D. Melb.; John Macpherson Harker, M.B. Lond.; Howell William Howell, M.D. Lond.; Robert Andrew Hunter, M.D. Manitoba; Evan Jones, M.B. Lond., L.R.C.P.; Prince Mohan Kaul, M.B. Punjab; George Durant Kersley, M.D. Camb., L.R.C.P.; Parshotam Lal Khosla, M.B. Punjab; Ranald Ian Macdonald, M.D. Dalhousie; Noel Francis Maclagan, M.B. Lond.; Donald Uvedale Owen, M.D. Liverp.; Arthur Ewart Parks, M.D. Toronto; William Phillips, M.B. Wales, L.R.C.P.; Wilfrid Michael Priest, M.B. Lond.; Cedric Barrington Prowse, M.B. Camb.; Thomas Henry Pullar, M.B. Sheffield; William Hutton Scobie, M.B. Glasg.; Ronald Bodley Scott, M.B. Oxf.; Frederick Harwood Stevenson, M.D. Lond.; Thomas Scott Stone, M.B. Lond.; Edward Gwynne Thomas, M.B. Lond.; Hubert Carey Trowell, M.D. Lond.; John Francis Varley, M.B. Camb.: and Joseph Viccars Walker, M.B. Birm.

Licences to practise were conferred upon 134 candidates (118 men and 16 women) who have passed the final examination of the Conjoint Board and have complied with the by-laws. The following are the names and medical schools of the successful candidates:—

G. Abbey, Leeds; P. H. Addison, St. Mary's; Monica H. Austin, King's Coll.; Margaret M. Baddeley, Liverp. and West London; T. A. Baldwin, Univ. Coll.; R. Barnes, M.B., Manch.; A. M. F. Batty and A. A. W. Beach, L.D.S., Birmingham; G. F. Benjamin, Liverpool; R. Bennett and J. G. Bentley, St. Bart.'s; A. Birnbaum, London; R. W. Blakey, Leeds; Joan C. D. Bolt, Royal Free; R. W. W. Bose, Camb. and London; F. C. Bourgault du Coudray, London; A. Bowen-Davies, Camb. and Guy's; W. D. Bower, Camb. and St. Thomas's; D. J. T. Brinkworth, Middlesex; J. Britanischski, M.D. Berlin, London; H. W. Burge, King's Coll.; R. J. Buxton, Camb. and St. George's; Jean W. D. Calman, Univ. Coll.; B. S. Carter, St. Thomas's; T. Chadderton, Manchester; H. F. Chard and H. K. Childs, St. Thomas's; M. M. Cohen, M.B., Manch.; D. D. Coleman, Coll. Med. Evang. and Charing Cross; D. M. Cooper, St. Thomas's; Esther Copperman and H. Creditor, Univ. Coll.; F. J. Curtis, Camb. and Guy's; J. E. Davenport, Camb. and London; J. Davies, Cardiff and London; D. J. Dawson, St. Bart.'s; H. De, Calcutta and St. George's; Ursula D'Monte, M.B. Bombay, West London; E. J. Dennison, St. Thomas's; L. S. De Silva, King's Coll.; M. E. Disney, London; A. G. Donald, Guy's; J. F. H. Eagles, Bristol; G. J. Evans, Guy's; J. C. G. Evans, St. Thomas's; L. H. Evelyn and E. D. Falconer, Guy's; S. T. Falla, London; Catherine F. Forrest, Royal Free; Getel Gimpelson, King's Coll.; I. H. Gosset, Oxford and St. Thomas's; E. N. Hailey, Birmingham; P. P. Halemani, Bombay and Middlesex; J. L. Hamilton-Paterson, Univ. Coll.; B. A. E. Harley, Middlesex; Freda M. B. Harmer, Royal Free; E. H. C. Harper, St. Thomas's; H. R. Hartnell and B. T. W. Harvey, London; P. G. F. Harvey, St. Bart.'s; Anne E. M. Herbert, King's Coll.; A. Hilmy, Cairo and St. Bart.'s; F. G. Holman, Guy's; J. M. Holmes, M.B., Leeds; R. A. Hooper and W. R. S. Hutchinson, Camb. and St. Thomas's; L. G. Irvine, Camb. and London; L. S. Jaikaran, London; A. G. Johnson, Guy's; E. G. Jones, Cardiff and London; Grace M. Jones and Nellie E. Jones, Cardiff; S. O. Jones and G. E. Kerr, King's Coll.; H. B. D. Kettlewell, Camb. and St. Bart.'s; F. B. Khambatta, Bombay and West London; F. R. Kilpatrick, Cape and Guy's; B. H. Kirman, King's Coll.; S. B. Lavine, Jefferson and Middlesex; J. A. E. Lawn, St. Bart.'s; M. Lee, St. Thomas's; Beatrice Lewis, Univ. Coll.; M. B. Lindsay, St. Thomas's; A. E. Locke, Guy's; Madeleine Lorsignol, Royal Free; J. F. Lown, Camb. and St. Bart.'s; B. McArdle, Guy's; J. O. McDonagh, Univ. Coll.; B. M. McOwan, St. Bart.'s; G. A. Mais, St. Thomas's; S. O. Massey, Middlesex; S. F. Mitchell, Univ. Coll.; G. R. Morgan, R. J. G. Morrison, and L. R. Norsworthy, St. Bart.'s; Josie M. Oldfield, Royal Free; C. V. Oldroyd, Manchester; J. P. O'Shea, Guy's; E. D. Page, St. Mary's; E. B. Pawson, Camb. and St. Bart.'s; K. M. A. Perry, Camb. and London; A. G. Potts, Guy's; L. E. J. Poulier, Ceylon and St. George's; G. A. Ransome, St. Bart.'s; P. B. Rao, Madras and London; F. W. Roberts, Middlesex; H. J. M. Robinson, Camb. and St. George's; A. H. W. Roffey, King's Coll.; R. D. Roper, Camb. and St. Bart.'s; R. H. Rushton, King's Coll.; A. G. Salaman, Camb. and London; A. W. Sampey, London; S. S. Selladurai, Ceylon and Middlesex; [illegible]ray, Middlesex; F. W. Shepherd, B.Chir. Camb., St. [illegible]. W. Smith, Guy's; G. S. Steele-Perkins, Camb. and Guy's; K. W. Stroude, M.B., Liverp.; G. H. Stuart, Univ. Coll.; T. V. Tattersall, Camb. and Manchester; W. E. Thomas, Cardiff; R. E. S. Turner, Oxford and St. Bart.'s; H. G. Ungley, M.B., Durh.; E. J. Walsham, St. Thomas's; S. P. Wanchoo, Punjab and Middlesex; G. L. Ward and C. J. T. Watson, Camb. and St. George's; E. E. S. Wheatley, St. Thomas's; W. H. Whiles, King's Coll.; I. Whittington, St. George's; J. Wilton, Witwatersrand and St. Mary's; V. F. Forbes Winslow, St. Bart.'s; E. J. S. Woolley, St. George's; and Freida Yarmalinsky, Univ. Coll.

The Milroy lectures, on Pneumococcal Infections, will be delivered by Dr. Robert Cruickshank at the College on Feb. 23rd, 28th, and March 2nd; the Goulstonian lectures, on the Physiology of the Gall-bladder and its Functional Abnormalities, by Dr. Charles E. Newman, on March 7th, 9th, and 14th; and the Lumleian lectures, on Complications of the Specific Fevers, by Dr. Charles R. Box, on March 16th, 21st, and 23rd.

## University of London

At a meeting of the senate held on Jan. 25th Mr. L. N. G. Filon, F.R.S., Goldsmid professor of applied mathematics and mechanics in the university, was elected vice-chancellor for the remainder of the year 1932–1933, in the place of the late Mr. J. L. S. Hatton.

The degree of D.Sc. (in human anatomy) was conferred on Dr. Francis Davies, university reader in anatomy at King's College, and (in physiology) on Dr. E. W. Fish, assistant dental surgeon to the Royal Dental Hospital and St. Mary's Hospital. The title of reader in experimental physiology in the university was conferred on Dr. H. P. Gilding, senior lecturer in physiology at University College.

Among those appointed Fellows of King's College was Mr. Eardley Holland, obstetric and gynæcological surgeon to the London Hospital. Mr. Holland was a student of the College from 1898 to 1904, and subsequently tutor in obstetrics and assistant obstetric surgeon to King's College Hospital.

Six lectures on Chemical Structure and Pharmacological Action—a study of drugs with specific actions on nerve-endings—will be given in the physiology theatre of University College, Gower-street, W.C., by Mr. H. R. Ing, D.Phil., at 5 P.M. on Mondays from Feb. 13th.

## Scottish Conjoint Board

As a result of recent examinations of the conjoint examining board of the Royal Colleges of Physicians and Surgeons of Edinburgh and the Royal Faculty of Physicians and Surgeons of Glasgow the following candidates have been granted the diplomas of L.R.C.P. Edin., L.R.C.S. Edin., L.R.F.P. and S. Glasg.:—

L. R. L. Solomon, Louis Danzis, M. O. Luck, George Kwryan, R. H. Payne, M. G. Myers, N. C. Smiedt, I. S. Lipschutz, E. L. Mommen, G. L. G. Major, A. A. Khan, C. A. S. O'Doherty, Jacomina A. Richter, J. G. Harrison, Aminuddin Ahmed, D. H. McNamara, Robert Keay, W. J. Silva, K. K. Retnam, Janet R. Hardy, C. W. Cook, Stewart McCallum, Noah Philips, A. L. Kahn, and J. C. T. Reid.

## Dr. Alfred Cox Testimonial Fund

The secretary to the committee organising this Fund, Dr. Rowland Fothergill, has issued the final balance-sheet giving the details of the artist's fee, the cost of the appeal, and the cost of the presentations. Reproductions of the portrait will not be undertaken, and subscribers have had returned to them the amount forwarded in respect of copies, but any subscriber who has not received back his remittance for a copy should communicate with Dr. Fothergill, 16, Brunswick-place, Hove, Sussex.

## British College of Obstetricians and Gynæcologists

A quarterly meeting of the council was held on Jan. 23rd, with Dr. J. S. Fairbairn, the president, in the chair. Dr. Henry Russell Andrews was elected a vice-president of the college, and Dr. Arthur E. Giles was elected honorary librarian. It was decided that the annual general meeting of the college should be held on April 5th in Birmingham, for the convenience of those attending the Congress of British Obstetrics and Gynæcology.

The following were formally admitted to the Fellowship: Cedric Lane-Roberts, Louisa Martindale, James Henry Drew Smythe; and the following to the Membership: Eric Arthur Gerrard, George Frederick Gibberd, Edith Hall, Alan Morris Johns, Richard Glyn Maliphant, Ellen Douglas Morton, William Joseph Rawlings, William Foster Rawson.

The following, having fulfilled the requirements of the by-laws, were elected to the Membership and will be admitted at the annual general meeting: William Saxon Barton, Alexander Broido, Jajneswar Chakravarti, Richard Victor Dowse, Wallace Freeborn, John Sydney Green, David McKay Hart, William Charles Wallace Nixon, James Vincent O'Sullivan, Robert Watson, Ralph Kuper White, Nellie Wilkes.

Dr. John Harry Hebb, C.B.E., has been appointed Director-General of Medical Services in the Ministry of Pensions, in succession to Sir Lisle Webb. Dr. Herbert Lightstone, D.S.O., will succeed Dr. Hebb as Deputy Director-General.

### Society of Chemical Industry

At a meeting of this society to be held at 8 P.M. on Monday, Feb. 6th, in the Chemical Society's Rooms, Burlington House, London, W., Dr. E. C. Dodds, Courtauld professor of biochemistry in the University of London, will read a paper on Hormones.

### St. George's Hospital

The medical school committee will hold a post-graduate course for old St. George's men from noon on Friday, Sept. 29th, to noon on Monday, Oct. 2nd, 1933, and a small committee has undertaken the arrangements. No details have been worked out yet, but the course will consist of lectures and demonstrations, chosen to meet the needs of general practitioners. The course will be followed on Monday, Oct. 2nd, by the general meeting of the school for the annual prize-giving and dinner, and a week-end ticket will cover the programme.

Ideas and suggestions from old St. George's men should be sent to Dr. Anthony Feiling, dean of the medical school.

### Research Fund for Mental Disorders

Through the medium of the council of the British Medical Association, Mrs. R. G. Burden, of Clevedon Hall, Clevedon, Somerset, warden of the National Institutions for Persons Requiring Care and Control, has announced her desire of making available for the encouragement and prosecution of research into mental problems and disorders the sum of £10,000. It is the donor's desire that the present director of medical services at Stoke Park Colony, Stapleton, Bristol (Prof. R. J. A. Berry), shall be in a position of responsibility with regard to the research work to be undertaken. In view of the national importance of such research active steps are being taken to form a committee of management and direction fully representative of the public interests.

### Fellowship of Medicine and Post-Graduate Medical Association

The fourth lecture in the series on practical problems in medicine and surgery will take place at 11, Chandos-street, Cavendish-square, London, W., at 4 P.M. on Tuesday, Feb. 7th, when Mr. Martin Oldershaw will lecture on the Significance of Bleeding as a Symptom in Gynæcology. A course of eight demonstrations on the Interpretations of Pyelograms is being given by Dr. G. R. Mather Cordiner, beginning on Wednesday, Feb. 8th, at 8 P.M. There will be a week-end course in physical medicine at the London Clinic and Institute of Physical Medicine on Saturday, Feb. 11th, and Sunday, Feb. 12th, when all-day instruction will be given. Forthcoming courses include an evening course at the National Temperance Hospital, Feb. 20th to March 17th; a course in medicine, surgery, and the specialties at the Prince of Wales's Hospital, Feb. 20th to March 4th; and in practical pathology at the Brompton Hospital, Feb. 20th to March 4th. All courses, lectures, and demonstrations arranged by the Fellowship are open only to members and associates. Further particulars may be had from the Secretary of the Fellowship at 1, Wimpole-street, London, W. 1.

### Grants for Research

The trustees of the Ella Sachs Plotz Foundation are anxious to make known the resources of their fund for the advancement of scientific investigation. The Foundation seldom or never offers stipends to investigators, nor does it provide apparatus and materials which are ordinarily part of laboratory equipment. Its special purpose is to give grants for the purchase of apparatus and supplies needed for special investigations and for the payment of unusual expenses incidental thereto, including technical assistance. At present research is favoured which bears on medicine or surgery, and the trustees endeavour to assist workers in various countries who are trying to solve identical or related problems. Thus a considerable number of grants have been made in the last few years for studies on nephritis, while internal secretion and infection have also been favoured, though not to so great a degree. In the nine years of its existence the Foundation has made 179 grants, and investigators have been aided in 23 countries. The sums provided last year ranged from 100 to 500 dollars each. Applications for assistance during the coming year should arrive before May 1st, including statements about the nature of the research, the amount of money wanted, and the objects on which it will be spent. The secretary of the executive is Dr. Joseph C. Aub, Corliss P. Huntington Memorial Hospital, Boston, Mass., U.S.A.

### The New Freemasons' Hospital

The report for 1932 presented to the annual meeting last week stated that although the dedication-stone of the new building at Hammersmith was laid only in May last by the Duke of Connaught, and a new wing has since been decided upon, the whole of the structure has been completed, along with the lighting and warming, and the interior work is well on the way to completion. The scheme has twice been enlarged, first from 120 to 144, and now to 180 beds. During the past year 687 patients passed through the present hospital in the Fulham-road, 329 of whom were a charge on the Samaritan Fund, against the previous highest figure of 270. Of this number 13 were cases from overseas, one being sent by aeroplane from Khartoum for an urgent operation. Although there have been many extras in connexion with the new building, the contract figure of £313,000 has not been exceeded. The new building will, it is expected, be open for the reception of patients in September or October.

## Medical Diary

*Information to be included in this column should reach us in proper form on Tuesday, and cannot appear if it reaches us later than the first post on Wednesday morning.*

### SOCIETIES

ROYAL SOCIETY OF MEDICINE, 1, Wimpole-street, W.

TUESDAY, Feb. 7th.—5.30 P.M., ORTHOPÆDICS. (Cases at 4.30 P.M.) Demonstration. Mr. E. I. Lloyd: Tilting Table for a Hip Operation in Children. Cases. Mr. G. R. Girdlestone: 1 and 2. Unusual Abnormalities of the Body of the Vertebræ. Mr. J. M. Turner: 3. Dyschondroplasia. 4. Hæmophilia with Tuberculous Abscess in Abdominal Wall and Arthritis of Hip. 5. Hæmangiectatic Hypertrophy of the Calf. Mr. C. Lambrinudi: 6. Pes Calcaneus. Mr. Philip Glynn: 7. Fractured Neck of the Femur treated with Smith-Petersen Pins. Mr. A. Rocyn Jones: 8. Madelung's Deformity. Other cases will be shown. 8 P.M., PATHOLOGY. Laboratory Meeting at the Lister Institute of Preventive Medicine, Chelsea Bridge-road, S.W.

FRIDAY.—5.30 P.M., CLINICAL. (Cases at 4.30 P.M.) Cases. Mr. H. A. T. Fairbank: 1. Congenital Enlargement of One Arm. Mr. J. P. Lockhart-Mummery: 2 and 3. Epithelioma of Anus treated with Radium. Prof. H. H. Woollard: 4. Myeloma successfully treated by X Rays. Mr. Harold Edwards: 5. Œsophageal Pouch. Dr. H. S. Stannus: 6. Tumour of Neck. Dr. Francis Bach: 7 and 8. High Blood Pressure successfully treated. Dr. F. Parkes Weber: 9. Chronic Enlargement of Parotid Glands of 16 years' duration, with Xerostomia. 10. Chronic Enlargement of Parotid Glands with Intermittent Exacerbations. 11. Chronic Œdema of Legs, with large Turgid Hands. 12. Dystonia Musculorum, probably of Encephalitic Origin. Dr. H. V. Morlock: 13. Chronic Spontaneous Pneumothorax treated by Intrapleural Injection of Silver Nitrate. 14. Repeated Hæmoptysis caused by a Benign Bronchial Neoplasm. Dr. G. Gregory Kayne: 15. Retrosternal Goitre and Amyotrophic Lateral Sclerosis. Other cases will be shown. 8.30 P.M., OPHTHALMOLOGY. Papers. Miss Ida Mann: On Congenital Hyaline Membranes on the Posterior Surface of the Cornea. Mr. J. Cole Marshall: Safar's Method of Treatment of Detachment of the Retina with Diathermy. Mr. N. Bishop Harman: A New Hand Slit-lamp. Mr. J. Foster: Method of Transferring Scotomata and Fields from a Scotometer Chart to a Perimeter Chart.

SOUTH-WEST LONDON MEDICAL SOCIETY.

WEDNESDAY, Feb. 8th.—9 P.M. (at the Bolingbroke Hospital, Wandsworth Common), Dr. A. Moncrieff: Coughs in Childhood.

WEST KENT MEDICO-CHIRURGICAL SOCIETY.

FRIDAY, Feb. 10th (at the Miller Hospital), Mr. P. H. Mitchiner: Should One Wait and See in Acute Appendicitis?

MEDICAL SOCIETY OF INDIVIDUAL PSYCHOLOGY, 11, Chandos-street, Cavendish-square, W.

THURSDAY, Feb. 9th.—8.30 P.M., Dr. W. J. O'Donovan: Pathomimia Cutanea.

### LECTURES, ADDRESSES, DEMONSTRATIONS, &c.

ROYAL COLLEGE OF SURGEONS OF ENGLAND, Lincoln's Inn-fields, W.C.

MONDAY, Feb. 6th.—5 P.M., Mr. H. P. Winsbury-White: Paths of Infection between the Genital Organs and the Urinary Tract. (Hunterian lecture.)

WEDNESDAY.—5 P.M., Mr. Stanford Cade: Radiation Treatment of Cancer of the Mouth and Pharynx. (Hunterian lecture.)

FRIDAY.—5 P.M., Mr. H. J. Burrows: Tissue Culture: its Relationship to Surgical Pathology. (Hunterian lecture.)

FELLOWSHIP OF MEDICINE AND POST-GRADUATE MEDICAL ASSOCIATION, 1, Wimpole-street, London, W.

MONDAY, Feb. 6th, to SATURDAY, Feb. 11th.—ST. JOHN'S HOSPITAL FOR DISEASES OF THE SKIN, Leicester-square, W.C. Post-graduate Course in Dermatology. Afternoon instruction in the Out-patient Department daily; lectures at 5 P.M. on Tues. and Thurs.—ROYAL FREE HOSPITAL, Gray's Inn-road, W.C. Demonstration on Antenatal Diagnosis and Treatment, by Dame A. Louise McIlroy, 5 P.M. Frid.—CHELSEA HOSPITAL FOR WOMEN, Arthur-street, S.W. A Course in Gynæcology. Mornings and/or afternoons daily.—HOSPITAL FOR CONSUMPTION, Brompton, S.W. A Course in Diseases of the Chest. All day instruction given.—LONDON CLINIC AND INSTITUTE OF PHYSICAL MEDICINE, Ranelagh-road, S.W. Week-end Course in Physical Medicine, Sat., Feb. 11th and Sund., Feb. 12th. All day tuition.—MEDICAL SOCIETY OF LONDON, 11, Chandos-street, Cavendish-square, W. Lecture on the Significance of Bleeding as a Symptom in Gynæcology, by Mr. Martin Oldershaw, on Tues., at 4 P.M. (Free to Members and Associates of the Fellowship.)—FELLOWSHIP OF MEDICINE, 1, Wimpole-street, W. A Series of Demonstrations on the Interpretation of Pyelograms will be given by Dr. G. R. Mather Cordiner Wed. at 8 P.M., and Sat. at 3 P.M.

ROYAL NORTHERN HOSPITAL, Holloway-road, N. (Intensive Week.)

MONDAY, Feb. 6th.—2.15 P.M., Mr. F. D. Saner: Some Aspects of the Treatment of Acute Suppuration. 3.15 P.M., Mr. Frederick Roques: Anæsthetics in Obstetric Practice.

TUESDAY.—2.15 P.M., Mr. McNeill Love: Some Common Diseases of Joints. 3.15 P.M., Mr. Bruce Williamson: Demonstration of Cases (unselected).

WEDNESDAY.—2.15 P.M., Dr. Bernard Schlesinger: Chorea. 3.15 P.M., Dr. Henry C. Semon: The Drug Rashes.

THURSDAY.—2.15 P.M., Dr. A. Lisle Punch: The Value of Lipiodol in Diagnosis. 3.15 P.M., Mr. E. Wolff: Common External Diseases of the Eye.

FRIDAY.—2.15 P.M., Dr. G. R. Mather Cordiner: Radiological Dyspepsias. 3.15 P.M., Mr. E. I. Lloyd: The Semilunar Cartilages of the Knee.

MIDDLESEX HOSPITAL MEDICAL SCHOOL, Mortimer-street, W.

TUESDAY, Feb. 7th, and the following THURSDAYS and TUESDAYS at 5 P.M., Prof. Samson Wright: Physiology of the Nervous Systems (six lectures).

ST. MARK'S HOSPITAL, City-road, E.C.

THURSDAY, Feb. 9th.—4 P.M., Mr. C. N. Morgan: Surgical Anatomy of the Anal Canal and Rectum.

CENTRAL LONDON THROAT, NOSE, AND EAR HOSPITAL, Gray's Inn-road, W.C.

FRIDAY, Feb. 10th.—4 P.M., Mr. W. G. Scott Brown: Œsophageal Obstruction.

ST. PETER'S HOSPITAL FOR STONE, Henrietta-street, W.C.

WEDNESDAY, Feb. 8th.—3 P.M., Mr. F. J. F. Barrington: Treatment of Senile Enlargement of the Prostate.

LONDON SCHOOL OF DERMATOLOGY, St. John's Hospital, 49, Leicester-square, W.C.

TUESDAY, Feb. 7th.—5 P.M., Dr. H. C. G. Semon: Diseases of the Buccal Mucous Membrane.

THURSDAY.—5 P.M., Dr. H. Haldin-Davis: Pruritus. Prurigo. Lichenification.

ST. MARY'S HOSPITALS, Manchester.

FRIDAY, Feb. 10th.—4.15 P.M., Post-graduate Lecture (at the Whitworth-street West Hospital), Dr. Lacey and Dr. Addis: Antenatal Demonstrations.

LONDON JEWISH HOSPITAL, Stepney Green, E.

THURSDAY, Feb. 16th.—4 P.M., Mr. S. I. Levy: Lecture-Demonstration. Enlarged Prostate and its Treatment

KING'S COLLEGE HOSPITAL MEDICAL SCHOOL, Denmark-hill, S.E.

THURSDAY, Feb. 9th.—9 P.M., Dr. Graham Hodgson: The Radiology of the Accessory Nasal Sinuses.

ROYAL INSTITUTE OF PUBLIC HEALTH, Queen-square, W.C.

WEDNESDAY, Feb. 8th.—4.30 P.M., Dr. W. S. C. Copeman: A Scheme for Industrial Rheumatism.

SOUTH-WEST LONDON POST-GRADUATE ASSOCIATION.

WEDNESDAY, Feb. 8th.—4 P.M., Mr. Terence Millin: Endoscopic Diathermy as applied to the Obstructing Prostate.

GLASGOW POST-GRADUATE LECTURES.

TUESDAY, Feb. 7th.—3.30 P.M. (in the Faculty Hall, St. Vincent-street), Mr. A. MacLennan: Post-Hospital Treatment of Deforming Lesions.

WEDNESDAY.—4.15 P.M. (at the Royal Samaritan Hospital for Women), Dr. D. McIntyre: Gynæcological Cases.

## Appointments

LEWIS, IVOR, M.D., M.S. Lond., has been appointed Medical Superintendent of the North Middlesex Hospital.

RYCROFT, B. W., M.D., Ch.B. St. And., D.O.M.S., Ophthalmic Surg[illegible] 'Second) to the Royal Northern Hospital.

## Vacancies

*For further information refer to the advertisement columns*

*Archway Hospital, Archway-road, Highgate, N.*—Asst. M.O. £250.

*Ayr County Hospital.*—H.S. £150.

*Bath, Royal United Hospital.*—Two Surgeon Dentists.

*Birkenhead General Hospital.*—Hon. Orthopædic Surgeon. Res. Sen. H.S. At rate of £150. Also Second H.S., H.P., and Cas. O. Each at rate of £100.

*Birmingham, Canwell Hall Babies' Hospital.*—Res. M.O. At rate of £250.

*Birmingham, Selly Oak Hospital.*—Cas. O. At rate of £200.

*Bolingbroke Hospital, Wandsworth Common, S.W.*—Asst. Gynæcologist.

*Bootle General Hospital.*—H.P., Two H.S.'s, and Cas. O. All at rate of £150.

*Bradford Children's Hospital.*—H.S. £100.

*Bristol General Hospital.*—Hon. Asst. Phys.

*Cardiff City Mental Hospital, Whitchurch.*—Sen. Asst. M.O. £570.

*Colchester, Essex County Hospital.*—H.P. £150.

*Coventry, Coventry and Warwickshire Hospital.*—H.S. to Aural and Ophthalmic Depts. At rate of £125.

*Croydon County Borough.*—Asst. M.O.H. for Obstetrics. £750.

*Derby County Mental Hospital, Mickleover.*—Deputy Med. Supt. £600.

*East Ham Memorial Hospital, Shrewsbury-road, E.*—Hon. Surg. to Ear, Nose and Throat Dept. Also Res. M.O. At rate of £200.

*East Sussex Public Assistance Committee.*—District M.O. &c. for Hove, &c. £60. Also District M.O. for Aldrington, &c. £85.

*Edinburgh, General Board of Control for Scotland.*—Medical Deputy Commissioner. £726.

*Fulham Hospital, St. Dunstan's-road, Hammersmith.*—Asst. M.O. £250.

*Gloucester County and City Mental Hospitals.*—Jun. Asst. M.O. £350.

*Gravesend and North Kent Hospital.*—Jun. H.S. £100–£150.

*Grosvenor Hospital for Women, Vincent-square, S.W.*—Chief Asst.

*Hackney Hospital, High-street, Homerton, E.*—Res. Asst. M.O. £350.

*Hampstead General and North-West London Hospital, Haverstock-hill, N.W.*—Cas. M.O. for Out-patient Dept. At rate of £100.

*Hastings, Royal East Sussex Hospital.*—Sen. and Jun. H.S.'s. At rate of £200 and £150 respectively.

*Highgate Hospital, Dartmouth Park Hill, N.*—Asst. M.O. £250.

*Hospital for Sick Children, Gt. Ormond-street, W.C.*—H.P. and H.S. Each at rate of £100.

*Hull Royal Infirmary.*—Third H.S. At rate of £150.

*Ilford, King George Hospital.*—H.S. At rate of £100.

*India, Leprosy Hospital, Dichpali.*—M.O.

*Leeds General Infirmary.*—Radio-Surgical H.S. At rate of £100. Also Hon. Ophth. Surgeon.

*Leicester Royal Infirmary.*—Hon. Asst. Radiologist.

*London Hospital, E.*—Med. First Asst. and Reg. £300.

*London Jewish Hospital, Stepney Green, E.*—H.S. At rate of £100.

*London Lock Hospital, 91, Dean-street, W.*—Surg. Reg. £100.

*Manchester, Ancoats Hospital.*—Hon. Asst. Phys.

*Miller General Hospital, Greenwich-road, S.E.*—Hon. Asst. Physician.

*Ministry of Health, Whitehall, S.W.*—Vacancy. £833.

*New End Hospital, Hampstead, N.W.*—Asst. M.O. £250.

*Northwood, Mount Vernon Hospital.*—H.S. At rate of £150.

*Norwood Children's Hospital, S.E.*—Asst. M.O. £250.

*Nottingham General Hospital.*—H.S. At rate of £150.

*Paddington Green Children's Hospital, W.*—Hon. Surgeon to Ear, Nose and Throat Dept.

*Portsmouth, St. Mary's Hospital, &c.*—Third Asst. Res. M.O. £250.

*Royal Northern Hospital, Holloway, N.*—Obstetric H.S. At rate of £70.

*Royal Surgical Aid Society, Salisbury-square, E.C.*—Hon. Ophth. Surg. 50 guineas.

*St. Alfege's Hospital, Vanbrugh-hill, S.E.*—Res. Asst. M.O. £350.

*St. Benedict's Hospital, Church-lane, Tooting, S.W.*—Asst. M.O. £250.

*St. George-in-the-East Hospital, Raine-street, Wapping, E.*—Asst. M.O. £250.

*St. Giles' Hospital, Brunswick-square Camberwell, S.E.*—Res. Asst. M.O. £350.

*St. Luke's Hospital, Sydney-street, S.W.*—Asst. M.O. £250.

*St. Mary Abbot's Hospital, Marloes-road, Kensington, W.*—Temp. Asst. M.O. At rate of £250.

*St. Mary Islington Hospital, Highgate-hill, N.*—Res. Asst. M.O. £350.

*St. Mary's Hospital for Women and Children, Plaistow, E.*—Hon. Ophthalmic Surgeon.

*Stoke-on-Trent, North Staffordshire Royal Infirmary.*—Asst. H.P. £125.

*Sunderland, Royal Infirmary.*—Sen. H.S. £200.

*Swansea County Borough.*—Asst. M.O. £500.

*Victoria Hospital for Children, Chelsea, S.W.*—Temporary Phys. to Out-patients.

*West London Hospital, Hammersmith-road, W.*—Asst. Phys. for Diseases of Children.

*West Riding of Yorkshire C.C.*—School Medical Inspector. £500.

*Westmorland County Hospital, Kendal.*—H.S. At rate of £200.

The Chief Inspector of Factories announces vacancies for Certifying Factory Surgeons at Leeds (West), East Farsley, and at Tuxford (Nottingham).

# NOTES, COMMENTS, AND ABSTRACTS

## IONIZED IODINE

(THE MOLSON IONIZED IODINE COMPANY, 34B, GABRIEL'S-HILL, MAIDSTONE)

AN investigation of the properties of the preparation ionized iodine (Molson brand) has been undertaken by THE LANCET and the results are as follows:—

The composition of this solution as given on the label sent out on the bottles is—

| | Parts. | | Parts. |
|---|---|---|---|
| Iodine .. .. | 7·91 | Ammonium .. .. | 1·02 |
| Chlorine .. .. | 10·04 | Hydrogen .. .. | 0·05 |
| Potassium .. .. | 2·89 | Salicylic acid .. .. | 2·00 |
| Sodium .. .. | 1·16 | Chlorophenols .. .. | 2·00 |
| Magnesium .. | 0·76 | Total .. .. | 27·83 |

One part of this mixture is dissolved in 115 parts of 10 per cent. glycerol.

Quantitative analysis on the same basis gives the following results, a table calculated in percentages from the above figures being added for purposes of comparison.

| | Found. (Parts per cent.) | Calculated. (Parts per cent.) |
|---|---|---|
| Iodine .. .. | 0·2429 ..... | 0·2440 |
| Chlorine .. .. | 0·3117 ..... | 0·3240 |
| Potassium .. .. | 0·0920 ..... | 0·0910 |
| Sodium .. .. | 0·0366 ..... | 0·0362 |
| Magnesium .. .. | 0·0230 ..... | 0·0237 |
| Hydrogen .. .. | 0·0016 ..... | 0·0015 |
| Ammonium .. .. | 0·0315 ..... | 0·0318 |
| Salicylic acid .. | 0·0628 ..... | 0·0624 |
| Chlorophenols .. | 0·0621 ..... | 0·0624 |
| Total .. .. | 0·8642 | 0·8770 |

The solution is clear to the naked eye, microscopically, and under conditions of dark-ground illumination. Some of the constituents are in a colloidal form, and can be separated by the addition of alkali and subsequent acidification. Attempts to redissolve the precipitate thus formed, and at the same time maintain a neutral or faintly acid reaction, were not successful. The constituents which separate in this way are salicylic acid and chlorophenols; no attempt to separate and characterise the various chlorophenols present was attempted.

The preparation is isotonic, and the lowering of freezing point ($\Delta$) is $-0{\cdot}52^{\circ}$ C.

Ionized iodine is faintly acid in reaction, having a pH value of 2·5 and a titratable acidity equivalent to 12·35 c.cm. of N/10 caustic soda per cent.; 4·45 c.cm. of this is accounted for by the salicylic acid present, the remainder probably being in the form of mixed hydrochloric, hydriodic, hypochlorous, and hypoiodous acids. Calculated as hydrochloric acid, this figure would be equivalent to a concentration of hydrochloric acid of 0·0209 per cent., or approximately 0·06 per cent. of the ordinary concentrated solution.

Iodine is present to the extent of 0·0617 per cent. in a free state, whilst the total content is 0·2429 per cent. The difference between free and combined iodine (0·1812 per cent.) is in an inorganic state, there being none of this element in organic combination. The mode of combination is complicated, as chloro-iodine compounds, hydriodic acid, hypoiodous acid, iodic acid, and iodides can all be detected in the solution.

Chlorine is present in combination (1) with iodine, (2) as inorganic chlorides, (3) as hydrochloric acid, (4) in traces as hypochlorous acid and possibly as chloric acid or its salts, and (5) as cyclic organic compounds in the form of chlorophenols. The amount available as a free combining agent can be deduced from its action on unsaturated organic compounds and from the oxidising properties of the solution. Estimated in this way, the combining power was found to be equivalent to approximately 0·02 per cent. of free chlorine.

The preparation has a powerful oxidising action on both inorganic salts (such as ferrous salts) and on organic compounds, many dyestuffs for example being bleached. This action is due partly but not wholly to the free iodine present, as when this element is rendered inactive by the addition of sodium thiosulphate oxidising properties still remain. The total amount of oxygen available which can be accounted for by the free iodine is 0·0038 per cent., whilst experimentally there is available 0·0110 per cent. This difference is due to the various iodo- and chloro- acids mentioned above. On account of this powerful oxidising action the solution is corrosive when in contact with metals for any length of time, and after long periods it will destroy organic material such as cork, silk, and wool; for this latter action to occur, however, contact with the preparation must be for some weeks.

### ACTION ON PROTEIN

The solution does not precipitate blood albumin, globulin, or fibrinogen. Serous effusions, provided that they do not contain any mucus, are similarly not affected. Mucus is precipitated by the solution in its natural state, but not if it is previously rendered alkaline, the precipitating agent being the acid and not any of the other constituents. The free iodine present is slowly taken up by protein solutions (so that the mixture gradually loses most of its colour) to form additive compounds as the element cannot be recovered subsequently in an inorganic state. In the presence of weak alkali the iodine is very rapidly absorbed, and cannot be liberated again on subsequent acidification. If the preparation is left in contact with a protein such as gelatin, and is then poured off after the visible iodine is absorbed, it still possesses iodising and oxidising properties, so that when it is again placed in contact with gelatin, iodine (and chlorine) pass into the protein and combine with it as before until the whole of the available supply is used up. In a similar manner, if a slab of gelatin is placed in contact with the solution it will be stained with iodine to a depth proportional to the length of time of contact; for a considerable depth below the visible stain, however, both iodine and chlorine, as additive organic compounds, can be detected showing that iodo-chloro compounds of considerable penetrative power are present. These iodo-chloro compounds, which penetrate deeply in this manner, combine with the protein and are not recoverable wholly in the inorganic form in which they were originally present.

### ACTION ON RED BLOOD CORPUSCLES AND HÆMOGLOBIN

The effect on hæmoglobin solutions is to form a mixture of acid hæmatin and methæmoglobin. This change occurs when the concentration of the antiseptic in the solution exceeds 1·8 per cent., and takes place almost instantaneously when a concentration of 4 per cent. is reached. When whole blood (oxalated) is used this action is not observed until at least an equal volume of antiseptic is added and no decomposition of hæmoglobin takes place. This is due presumably to the fact that whole blood is sufficiently well buffered to overcome the free acid present in the antiseptic, whilst the protein combines with the free and combined iodine and chlorine and prevents their oxidative action on the pigment.

No hæmolysis of red blood corpuscles occurs even after 24 hours when washed cells are suspended in the undiluted antiseptic, but naturally takes place with solutions diluted with water, but not when normal saline is used. No alterations in the volume of the cells takes place under the same conditions, as determined by hæmatocrite measurements. When washed cells are used for these determinations an almost immediate conversion of their contained hæmoglobin as described above takes place, but this is not observed when whole (oxalated) blood is used.

### ACTION ON LEUCOCYTES

The shape and staining reactions, both of cytoplasm and nucleus of all types of white blood corpuscles, are not altered by contact with the antiseptic. This applies to blood films made and stained in the ordinary way with Leishman stain after treatment of the dried film for five minutes with the undiluted antiseptic, and also to films made from oxalated blood and blood taken directly into the solution and subsequently stained. Effects upon the opsonic index were not determined.

### BLOOD COAGULATION

Blood coagulation is not delayed, and the clot formed in the presence of the antiseptic possesses normal firmness and is normally retractile. This property was tested on oxalated blood with subsequent addition of an amount of calcium chloride sufficient to restore a normal ionic calcium content to the mixture. When mixed with an equal quantity of antiseptic clotting is delayed, but not more than in a control preparation in which normal saline replaced the antiseptic, the delay in both cases being due to dilution only. When only small quantities of antiseptic are used—e.g., up to one-tenth of the volume of the blood—no delay was observed. Similar tests on scarified areas of skin showed that the blood oozing through on to dressings moistened with ionized iodine clotted in a normal manner, and also that dressings applied in this way did not adhere to the wounds.

### DIFFUSION EXPERIMENTS

Diffusion across collodion membranes takes place readily, all the constituents passing through. When dialysed against water, inorganic chlorides and iodides can be detected on the other side of the membrane in from two to five minutes, depending upon the thickness of the membrane and its method of preparation—i.e., on its degree of porosity. Free iodine appears shortly afterwards, then chloro-compounds (inorganic), and, finally, the salicylic acid and chlorophenols; some retention within the membrane itself appears to take place with the latter compounds.

Diffusion across solid slabs of gelatin has been described above, where free iodine was shown to be retained whilst the other constituents diffused more readily. Similar experiments using omentum as a semipermeable, natural membrane showed that the free iodine was retained almost completely within the membrane, whilst the other constituents diffused through readily, the dialysate possessing antiseptic and oxidising properties, although these were considerably less than is the case with the undiluted product.

Rapid diffusion occurs in agar-agar plates; if a cotton-wool pad well soaked in the antiseptic and contained in the end of a short open glass tube is placed in the centre of a freshly inoculated Petri dish, diffusion towards the edge of the dish is sufficiently rapid to prevent the growth of organisms excepting perhaps at the extreme periphery. The rate of diffusion and the dilution taking place depend amongst other factors on the thickness of the agar plate, the percentage amount of agar present, and the amount of antiseptic used. The inhibiting action is not so marked when serum-agar is used, due probably to the fact that unsaturated compounds are present which remove the active iodine and chlorine by forming additive bodies.

### ANTISEPTIC POWER

Tested on *B. typhosus*, *Staphylococcus aureus*, and hæmolytic streptococci it was found that cultures of these organisms could be killed in 2½ minutes by the antiseptic diluted with an equal amount of water. About half of this antiseptic power is due to the free iodine present, as when this element is eliminated by the addition of sodium thiosulphate the above organisms required treatment with the undiluted antiseptic for 2½ minutes before their growth was inhibited. The presence of sterilised (boiled) fæces, blood-serum, or ascitic fluid does not materially affect the antiseptic properties of the preparation.

Control experiments with standard phenol solutions showed that the test organisms were killed in a similar period by 2½ per cent. (1 in 40) carbolic acid.

### LETHAL ACTION, AND ACTION ON MUCOUS MEMBRANES

Approximately 12 c.cm. per kilo of body-weight were found to be lethal when injected intravenously into rabbits. This action is due largely to the free iodine present; when this is removed by means of thiosulphate the preparation is toxic only in very large doses. When injected subcutaneously and intraperitoneally no toxic effects were observed with doses of 20 c.cm. per kilo of body-weight.

When applied continuously—e.g., for two hours—to mucous membranes, a coagulation of superficial mucus takes place, followed by histological evidence of increased glandular activity on the part of the mucus-secreting glands, and a mild polymorphonuclear leucocyte infiltration. No coagulation of tissues occurs. When applied for a short while only—e.g., for five minutes—no histological changes can be observed apart from the superficial coagulation of mucus. Repeated applications daily for a period of 14 days resulted in a mild submucous polymorphonuclear infiltration.

No changes in ordinary skin could be observed after daily applications for 14 days.

### DEODORISING ACTION

The preparation has a very marked deodorising action on such pathological material as fæces, pus, and decomposing urine.

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## RHEUMATISM AS A PUBLIC BURDEN

THE urgency of rheumatism as an industrial problem is the theme of a course of lectures now in progress at the Royal Institute of Public Health. Presiding at the first meeting, Sir Arthur Stanley said that he was glad to see that this matter was being taken up in academic circles. "Frankly," he said, "I had always thought that the medical profession was rather bored with it. But even the medical profession is moving with the times."

The first lecture was given on Jan. 18th by Dr. Langdon Brown, Regius professor of physic in the University of Cambridge, and the second on Jan. 25th by Dr. Fortescue Fox, chairman of the Ligue Internationale contre le Rheumatisme. The evolution of medicine, Dr. Fox said, seems to show that the problem of the epidemic is on its way to solution, that acute diseases are on the whole becoming less prevalent, and that we are reaching an era of chronic disease. This is a new problem, requiring new ways of approach; the objective is to save health rather than lives.

Reports submitted to the recent international rheumatism congress in Paris showed, said Dr. Fox, that the incidence of rheumatism is maximal in outdoor occupations, especially those with hard labour and exposure to cold. In Sweden, G. Kahlmeter found that the permanent invalidity considerably increased between 1918 and 1928, and now exceeds that from pulmonary tuberculosis. About 30,000 arthritic persons are now disabled in Sweden (6 per cent. of the population) at a charge upon the State more than twice as great as that for tuberculosis. J. van Breemen has analysed the occupations of 3000 patients in Holland (Amsterdam), and states that men in outdoor work have from three to five times more rheumatic abnormality than those occupied indoors. A report from Germany suggests that nearly 75 per cent. of industrial workers over 40 years of age suffer from some form of rheumatism. The report for the rheumatism committee in the Soviet Republics emphasised the great importance of chill, and gave figures showing a great reduction of invalidity in factories where this risk was minimised. An analysis of 19,000 cases treated at the chief spa hospitals in England confirmed the main conclusions

of the other reports: "Those most affected are, in men, the strenuous manual workers exposed to damp, and, in women, housewives and home workers."

Dr. Fox believes that many contributory factors are now taking a fixed place in the constellation "Rhuma." There is no disease, he thinks, for which a single cause is more impossible; at the same time it is common to find a dominant cause, and the vicious circle can be broken only at this point, and in favouring circumstances. He attributed much importance to the production of chronic chill by repeated exposure to uncompensated cold. Rheumatoid, the "British arthritis," was often connected with chill; osteo-arthritis, on the other hand, was related to mechanical trauma (injury).

Further lectures in the series are being given by Dr. C. W. Buckley, Dr. W. S. C. Copeman, and Miss Margaret Edminson. Particulars may be had from the institute, 23, Queen-square, London, W.C.1.

## INSTRUCTION ON NURSING PROCEDURES

THERE are many procedures on the borderland between medicine and nursing of which the doctor is often content to assume that the nurse has mastered the practical details, and thus issues his orders in general terms. This detached attitude may have its dangers, for not all private nurses have been recently trained. It is wise to be prepared to give precise instructions. An extraordinarily comprehensive little manual,[1] compiled primarily for practising nurses, will meet a need which the doctor may have felt also. Its author, Miss Powell, better known as Sister Hope, of the medical side of St. Bartholomew's Hospital, has provided a concise summary of the requirements of almost every medical emergency, and has included notes on the dietaries appropriate for various conditions. Miss Powell does not advocate any procedure, but mentions the occasion for which it may be ordered, and then tells the nurse exactly how to carry it out. Her style is simple, direct, and vivid, and her little book will be more often consulted by doctors and nurses than are many weighty tomes.

## HOSPITAL LIBRARIES

ON Jan. 25th a well-attended meeting, organised by the London and Home Counties Branch of the Library Association, on "Hospital Libraries" was held in the Nurses' Home of the Middlesex Hospital. Miss Frost, librarian to the public library at Worthing, presided. Mrs. R. Beddington, hon. librarian to the Middlesex Hospital, spoke of the system employed in hospital libraries. The Middlesex could claim to be one of the first hospitals to have a patients' library, since one had existed there before the war. As the result of many years' experience she emphasised the need for the right type of librarian and for a simple system of handling books. "In the past," she said, "hospital libraries were suffered to exist; now they are looked upon as part of the curative work of the hospital." Mrs. Richmond said that at Guy's no patients' library had existed before 1931; now there were eight librarians, 4800 books, and four trolleys. The wards were visited three times a week. In the paying wards a charge of 2*d*. a volume was made. Special facilities were given for the nurses to use the library. In most hospitals librarians worked in the afternoons, but at Guy's the routine of the distribution fitted in better with the routine work of the wards if it was done in the mornings. Mr. Bedwell, house governor to King's College Hospital, said that historically hospital libraries could be traced to the time when hospitals were monastic foundations, the monks being concerned with the advancement of learning equally with the care of the sick. In England St. Bartholomew's was the foremost example; the present library of the medical school of that hospital was developed from the library mentioned in the records of 1667. In England he knew of no medical school directly concerned with the provision of books for patients, but in the Royal Victoria Hospital, Montreal, a library was staffed and largely financed by women medical students, and was available for the administrative staff as well as for the patients. Mr. Bedwell laid stress on the importance of libraries for nurses and on the greater possibilities of library service in hospitals. Mr. Bolton described the plan in force at the Watford Peace Memorial Hospital. A small stock of public library books was kept in that hospital and was renewed when necessary. In this way a high standard of books in good condition was maintained; the work was carried out by ex-library assistants.

[1] Practical Preparations. By N. W. Powell. London Faber and Faber, Ltd. Pp. 206. 2*s*. 6*d*.

## THE YOUNG AERONAUT

Dr. Killick Millard, the well-known medical officer of the city of Leicester, has taken to flying at the age of 62, with such a rejuvenating effect upon himself that he has publicly recommended doctors to prescribe flying for their patients, as a substitute for sending them abroad. He holds that flying might be an ideal treatment for people in need of a change, while in his view this method of transit has become practically safe if only plain aeronautics are indulged in.

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HORMONES AND MENSTRUATION.—In a leading article with this title in our issue of Jan. 21st, reference was made to a report on a synthetic œstrus-exciting compound by Mr. J. W. Cook, D.Sc., Prof. E. C. Dodds, and Mr. C. L. Hewett. This report appeared in *Nature* of Jan. 14th (p. 56), and not Jan. 7th as stated in our article.

# Births, Marriages, and Deaths

## BIRTHS

HALL.—On Jan. 25th, at Wilbraham-place, S.W., the wife of Dr. J. H. Hall, of a daughter.

HOSFORD.—On Jan. 28, at "Cairnton," Stormont-road, Highgate, the wife of R. W. P. Hosford, F.R.C.S., of a son.

LLOYD.—On Jan. 25th, at Bentinck-street, W., the wife of W. Ernest Lloyd, M.D., of a son.

LYNN.—On Jan. 25th, in London, the wife of Lieut.-Colonel Rigby Lynn, D.S.O., I.M.S., of a son.

SCHNEIDER.—On Jan. 27th, at Southampton, to Dorothy (née Green), wife of John Erhard, M.B., B.S. (Lond.)—a daughter.

## MARRIAGES

KERR—NEVIN.—On Jan. 25th, at Carr's Lane, Birmingham, James Arthur Kerr, B.Sc., M.D., D.P.H., of Glasgow, to Catherine Dorothea Nevin, B.A., younger daughter of the late Dr. A. M. Nevin, of Sparkhill, Birmingham.

LAWSON—EVANS.—On Jan. 24th, at St. Paul's, Onslow-square, William Thomson Lawson, M.B., Llangadock, to Phyllis Marguerite, only daughter of the late H. Dawkin Evans, Esq., and of Mrs. Evans, Iscoed, Ferryside.

## DEATHS

COWEN.—On Jan. 23rd, in London, Edward James Cowen, M.B., T.C.D., aged 78.

ELPHICK.—On Jan. 27th, at Newton House, Kirkcaldy, Harry Noel Keyes Elphick, M.C., B.A. (Oxon.), M.R.C.S., L.R.C.P. Lond., aged 36 years.

EVANS.—On Jan. 24th, suddenly, at The Cottage, Lansdowne-road, Worthing, Thomas Evans, M.D., late Medical Superintendent, Archway Hospital, Highgate.

FLANEGAN.—On Jan. 22nd, Henry James Flanegan, F.R.C.S., of "The Grange," Wellesley-road, Clacton-on-Sea, and Eyrecourt, Co. Galway.

JONES.—On Jan. 27th, at Penderi, Creigiau, Cardiff, William Black Jones, M.D., J.P., formerly of Builth Wells, aged 68.

MARTIN.—On Jan. 28th, at George-road, Edgbaston, Birmingham, Christopher Martin, M.B., C.M., F.R.C.S.

OWENS.—On Jan. 29th, at Longstratton, Norfolk, Charles Arthur Owen Owens, M.D., V.D., J.P., in his 88th year.

SORAPURE.—On Jan. 29th, in a nursing-home at Bournemouth, Victor Edgar Sorapure, M.B., F.R.C.S. Edin., of 47, Wimpole-street, London.

TAYLOR.—On Jan. 29th, in Dublin, Sir William Taylor, Regius Professor of Surgery in Dublin University, aged 61.

WALL.—On Jan. 28th, Maximilian Christian Wall, M.R.C.S.Eng., of Fore-street, Edmonton, N.

*N.B.—A fee of 7s. 6d. is charged for the insertion of Notices of Births, Marriages, and Deaths.*